DOES NOT CIRCULATE

Vol. 38 OCTOBER, 1955 No. 4

SURGERY

A Monthly Journal Devoted to the Art and Science of Surgery

UNIVERSITY OF MICHIGAN
OCT 18 1955
HOSPITAL LIBRARY

EDITORS

ALTON OCHSNER, New Orleans OWEN H. WANGENSTEEN, Minneapolis

ASSOCIATE EDITORS

ALFRED BLALOCK, Baltimore I. S. RAVDIN, Philadelphia
JOHN R. PAINE, Buffalo WARREN H. COLE, Chicago

Original Communications

Nutrition in Gastric Surgery With Particular Reference to Nitrogen and Fat Assimilation. Hushang Javid, M.D., Ph.D., Chicago, Ill. ---------- 641

Malignant Melanoma. Robert H. Lund, M.D., and Menard Ihnen, M.D., St. Louis, Mo. ---------- 652

An Improved Technique for an Adequate Radical Groin Dissection for Malignancy. Stephen L. Gumport, M.D., and Herbert Willy Meyer, M.D., New York, N. Y. 660

The Use of a Triple-Lumen Single-Balloon Tube in the Diagnosis and Treatment of Massive Upper Gastrointestinal Hemorrhage. Marvin M. Nachlas, M.D., Boston, Mass. ---------- 667

Dissection of the Hepatic Pedicle and Retropancreaticoduodenal Areas for Cancer of the Stomach. Stuart W. Arhelger, M.D., Paul H. Lober, M.D., and Owen H. Wangensteen, M.D., Minneapolis, Minn. ---------- 675

A Method for Total Removal of Bile Duct Calculi. Julian A. Sterling, M.D., Sc.D., Philadelphia, Pa. With the Technical Assistance of Bernard Diamond, A.B., Ong Tiong-Giok, M.D., and Harold Furman, M.D. ---------- 679

Thermal Burns in Man. IV. Body Weight Changes During Therapy. James D. Hardy, M.D., Eugene Jabbour, M.D., John R. Lovelace, M.D., William A. Neely, M.D., and Frank C. Wilson, Jr., M.D., Memphis, Tenn. ---------- 685

Thermal Burns in Man. VII. "Insensible Fluid Loss." James D. Hardy, M.D., William A. Neely, M.D., and Frank C. Wilson, Jr., M.D., Memphis, Tenn. ---- 692

(Contents continued on inside back cover)

Published by THE C. V. MOSBY COMPANY, St. Louis, U. S. A.

Copyright 1955 by The C. V. Mosby Company

SURGERY

EDITORIAL BOARD

JOHN ADRIANI, New Orleans
ARTHUR W. ALLEN, Boston
EDWARD D. CHURCHILL, Boston
FREDERICK A. COLLER, Ann Arbor
MICHAEL DE BAKEY, Houston
CLARENCE DENNIS, Brooklyn
LESTER R. DRAGSTEDT, Chicago
ROBERT E. GROSS, Boston
EMILE F. HOLMAN, San Francisco
J. ALBERT KEY, St. Louis
JOHN J. MORTON, Rochester, N. Y.
CARL MOYER, St. Louis
THOMAS G. ORR, Kansas City, Kan.
GEORGE T. PACK, New York
WILDER G. PENFIELD, Montreal
WILLIAM F. RIENHOFF, JR., Baltimore

COMMITTEE ON PUBLICATIONS

J. Garrott Allen
Chicago, Ill.

William A. Altemeier
Cincinnati, Ohio

Isaac A. Bigger
Richmond, Va.

J. Barrett Brown
St. Louis, Mo.

Alexander Brunschwig
New York, N. Y.

O. T. Clagett
Rochester, Minn.

Warren H. Cole
Chicago, Ill.

C. D. Creevy
Minneapolis, Minn.

Daniel C. Elkin
Atlanta, Ga.

Frank Gerbode
San Francisco, Calif.

Charles W. Mayo
Rochester, Minn.

H. Thomas Randall
New York, N. Y.

Leo G. Rigler
Minneapolis, Minn.

Maurice B. Visscher
Minneapolis, Minn.

A. Earl Walker
Baltimore, Md.

James C. White
Boston, Mass.

Philip D. Wilson
New York, N. Y.

Vol. 38, No. 4, October, 1955. Surgery is published monthly by The C. V. Mosby Company, 3207 Washington Blvd., St. Louis 3, Mo. Subscription price: United States, $14.00 a year; Canada, $14.50; Foreign, $15.50. Entered as Second-Class matter January 20, 1937, at the Post Office at St. Louis, Missouri, under the Act of March 3, 1879. Printed in the U. S. A.

SURGERY

VOL. 38 OCTOBER, 1955 NO. 4

Original Communications

NUTRITION IN GASTRIC SURGERY WITH PARTICULAR REFERENCE TO NITROGEN AND FAT ASSIMILATION

HUSHANG JAVID, M.D., PH.D., CHICAGO, ILL.

(From the Department of Surgery, University of Illinois College of Medicine)

THE problem of nutritional disturbance following operative procedures upon the stomach has stimulated discussion for many years. A vast amount of information concerning the various physiologic effects of gastric procedures is available in the literature. Several authors[1, 2, 8, 54] have commented upon the extreme malnutrition present following total gastrectomy. Everson,[21] in his animal experiments and case studies, has shown conclusively that nutritional disturbances occur following total removal of the stomach. Evidence of faulty nutrition has also been observed in partial gastrectomy and other operative procedures upon the stomach. In our search of the literature, however, we were unable to find an experimental evaluation of fat and nitrogen assimilation following partial gastrectomy, gastroenterostomy, vagotomy, gastrectomy combined with vagotomy, and gastroenterostomy with vagotomy.

It is the purpose of this study to investigate the effects of these various procedures upon fat and nitrogen metabolism. Metabolic studies were conducted on normal dogs for two seven-day periods, in order to determine normal percentage values of ingested fat and nitrogen excreted in the feces. These animals were then subjected to the various gastric procedures mentioned previously. Fat and nitrogen intake-excretion studies were repeated on the operated dogs after complete recovery. Our results indicated that gastrectomy combined with vagotomy produces the greatest loss of fat and nitrogen in the feces.

REVIEW OF THE LITERATURE

Partial gastrectomy has been considered responsible for the loss of weight and diarrhea, which result in nutritional failure. Studies[2, 9, 11, 24, 31, 33, 39, 44, 46] made on patients following subtotal gastrectomy revealed that a significant number of patients were unable to maintain their weight. Ivy, Grossman, and Bachrach[33] compiled from the literature the results of 864 cases in which weight be-

Received for publication Dec. 22, 1954.

fore and after subtotal gastrectomy for peptic ulcer had been compared. In 42 per cent of these patients the postoperative weight was less than the normal preoperative weight.

In a recent study Bell and his co-workers[6] reported 154 cases of gastric resection for peptic ulcer. Of these, 103 were available for detailed study. Postoperatively, 57.9 per cent of these patients did not gain weight; some lost several pounds. Browne and McHardy[9] reported a loss of weight in 66 per cent of the gastrectomy cases. Additional evidence of impaired assimilation of fat and nitrogen following subtotal gastrectomy has been obtained from macroscopic and microscopic examination of the feces. Lublin,[34] for example, reported excessive amounts of fat, connective tissue, and muscle fibers in the feces of 5 of 9 patients studied after partial gastrectomy. Bohmansson[7] observed similar findings in the stools of 11 of 34 patients following this operation. Others have reported changes in the appearance of the feces[15, 16] after subtotal gastrectomy.

Wollaeger and co-workers[55, 58] became interested in the study of fecal fat and nitrogen in postgastrectomy patients because some of the patients complained of diarrhea and failure to gain weight. Examination of the stools in these patients indicated excessive loss of fat. Wollaeger's studies indicate that the amount of fecal fat increases following gastrectomy and is more pronounced in patients showing digestive symptoms and failure to gain weight. In 6 patients having mild or no digestive symptoms, the average loss of fat in the feces ranged from 6.5 to 9.5 per cent of ingested fat, while in 8 patients with severe gastrointestinal disturbance, the range was 9.1 to 45.2 per cent.

According to Wollaeger, indirect evidence of a possible disturbance in digestion and absorption is also obtained by noting the incidence of diarrhea in subjects who had undergone this type of operation. Meyer, Brams, and Graves[36] found diarrhea a postoperative complaint in 1 out of 12 cases in which the Billroth I operation had been performed. Hedlund[28] reported the occurrence of diarrhea in 1.5 per cent of his patients after partial gastrectomy. In Lublin's series of 11 patients who complained of untoward symptoms long after operation, 5 had diarrhea, 3 intermittently and 2 continuously. Gordon-Taylor and associates[26] reported 6 of 52 patients with postoperative diarrhea. They performed quantitative analyses of the fecal fat in 33 patients. Their results revealed high fat output in the feces; one-third had a higher output than the upper limit of normal. The highest fat value was 66 per cent of the dried weight of the stool. On microscopic examination of the feces excessive amounts of undigested muscle fibers, connective tissue, and fat were found in some cases. Von Haberer[48] also mentioned that patients who had undergone Billroth II operations had a tendency toward diarrhea and fatty stools.

Intake and excretion studies on subtotal gastrectomy were carried out by Gavrila and Danicico[25] on 13 patients who had had gastric resection, 12 of which were of the Billroth II type. The results of these investigations are similar to other studies. These investigators found an increased loss of fat in the feces in all of their patients. The loss of fat ranged from 6 to 46 per cent of the ingested fat. (The dietary intake of fat was more than 85 Gm. daily in every case.) They reported an excessive loss of nitrogen in 8 of the 13 cases.

The reason for the excessive loss of fat and nitrogen in the feces of patients following subtotal gastrectomy is as yet not completely understood. No doubt there are many factors responsible for this disturbance, since partial removal of the stomach is detrimental to the gastrointestinal physiologic processes. Wollaeger listed four important factors: (1) rapid emptying of the stomach; (2) "intestinal hurry"; (3) diminished flow of pancreatic secretions or bile due to impairment of the mechanism of stimulation of such flow; and (4) imperfect mixing of food with digestive ferments because of the shunting of gastric contents away from the duodenum.

Other investigators[20, 29, 40, 44] have also reported that similar factors are responsible for faulty digestion and absorption.

Vagus Section.—This has been the subject of extensive studies for the past decade. Little information has been available on the effect of vagotomy on nutrition until recent experiments were conducted by Welbourn, Hallenbeck, and Bollman.[52] In this study, fat intake-excretion studies were conducted and the results in four dogs revealed no significant change in the amount of fat excreted. The average in these animals was 2.6 per cent as compared with the control group average of 2.7 per cent.

Studies of Fox and Grimson,[22] however, revealed that in patients who have digestive complaints following vagotomy fat absorption is defective. In 9 patients studied, 8 had an absorption of less than 90 per cent, while all of the patients in the control group had an absorption higher than 90 per cent. The average fecal output for the vagotomy group exceeded, in bulk, that of the other patients.

Beal and Dineen,[5] in a study of their vagotomy patients, emphasized the occurrence of diarrhea after vagus section. Thirty-eight per cent of Miller's[37] patients had diarrhea. Grimson and associates[27] also reported an incidence of 30 per cent (11 patients of 36) with mild periodic diarrhea. A postoperative loss of weight was reported in 10 of 36 cases.

The cause of diarrhea following vagotomy is not as yet fully understood. Dragstedt[15] felt that anacidity of the stomach rendered the patient more susceptible to diarrhea of bacterial origin. Walters[50] has commented that jejunitis, which he found in some cases, might be a contributing factor. He stated that 5 patients in 69 had severe diarrhea. Gastric analysis in these patients revealed free acidity from 15 to 50 units after histamine. Changes in the motility of the small intestine has also been considered an important factor. According to the studies of Fox and Grimson, high fecal output of fat may be a responsible cause of diarrhea.

Gastroenterostomy and Vagotomy.—This procedure has been recognized as a better operation for peptic ulcer than vagotomy alone. This operation has gained increasing popularity in some clinics.[12, 14, 16, 27] Edwards and associates[19] reported satisfactory results in about 90 per cent of their cases; excellent results were noted in 60 per cent of the cases. Mild symptoms, such as fullness, inability to gain weight, and diarrhea were reported by the remaining patients. Crile and co-workers[14] reported 69 per cent excellent and 21 per cent

satisfactory results. With the combined procedure, Dragstedt's[16, 17] results are outstanding, being 90 per cent excellent. In his work, Walters[50] reported 75 per cent excellent results. An extensive study made by the Vagotomy Committee of the Gastro-enterological Association on 1,046 patients following vagotomy and gastroenterostomy revealed an excellent result in 93.2 per cent. Postoperative complaints such as diarrhea and other gastrointestinal symptoms have been reported following this combined procedure. Grimson and co-workers[27] reported diarrhea as a complication in six cases of 28. Collins's[12] studies in 95 cases of vagotomy with gastroenterostomy disclosed a loss of weight in 8 patients, three of whom were on a weight-reducing diet. According to the information available in the literature, it seems that vagotomy combined with gastroenterostomy is much less disturbing to the nutrition than gastric resection. Detailed fat and nitrogen balance studies are, however, needed to clarify this point.

Vagotomy combined with gastric resection has been advocated in recent years, in order to decrease the incidence of ulcer recurrence. Colp and associates[13] have reported 40 cases of gastrectomy with vagotomy for peptic ulcer with no incidence of recurrence. In a similar study of 46 cases of subtotal gastrectomy, one recurrence was noted. The night secretion studies in the gastrectomy-vagotomy group revealed achlorhydria in 19 of the 20 patients tested, as compared to 14 out of 21 cases following subtotal gastrectomy alone. There is, however, no mention of the nutritional status of these patients after the operation. A mild diarrhea was noted most commonly in the vagotomy-gastrectomy group. Dragstedt[15] commented that the combination of vagotomy with gastrectomy is unwarranted, since a large number of patients will lose an important digestive organ unnecessarily.

The information presented in the literature points to nutritional disturbances following these various operative procedures upon the stomach. Conclusive experimental evidence is needed, however, to clarify the degree of this disturbance in specific gastric operations. For this reason experimental studies as described below have been conducted in animals following various types of operations, with the hope of obtaining fundamental data and clarifying the indications for the various types of operations.

EXPERIMENTAL METHOD

Adult mongrel dogs were used for this investigation. Intake-excretion studies were accomplished on 31 normal dogs. The metabolic studies consisted of two seven-day periods. The animals were then divided into five groups; each group was subjected to a different gastric operation. In the vagotomy group completeness of vagus section was judged by a carefully made insulin test. Postoperatively, fat and nitrogen intake-excretion studies were determined and observations were made as early as six weeks and as late as six months after surgery. In the control group of 31 dogs the percentage value of ingested fat which was excreted in the feces ranged from 1.6 to 22.7 per cent, with an average of 5.04 per cent. The percentage of nitrogen excreted in the same group ranged from 1.6 to 35 per cent, with an average of 13.6 per cent. The dogs were given daily diets calculated and chemically analyzed to furnish 80 calories per kilogram of body weight and containing 5 Gm. of protein and 4.0 Gm. of fat per kilogram of body weight. Carmine markers were used to demarcate

the fecal collection periods. The feces for each period was mixed in a Waring blender and fat and total nitrogen were determined on aliquot samples. Nitrogen was determined with Hoffman's[30] modification of the Koch and McMeekin direct nesslerization micro Kjeldahl method, and fat excretion was determined using the Soxhlet method.

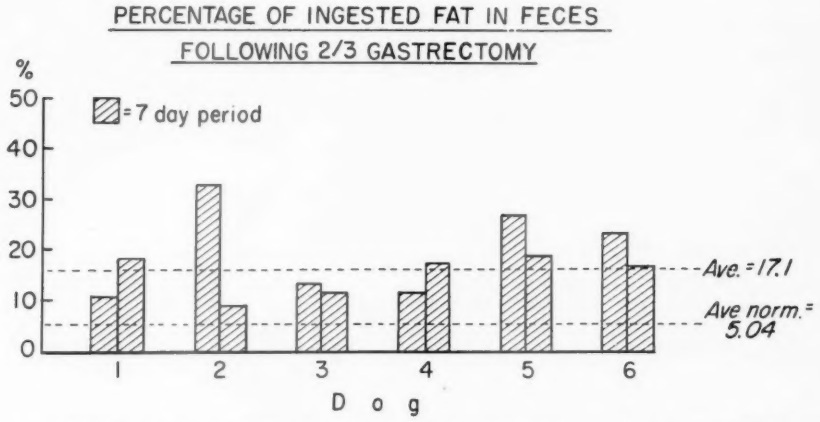


Fig. 1.—Metabolic studies following two-thirds gastrectomy. Two seven-day periods were observed in each of the six dogs. Average percentage of ingested fat excreted in the feces preoperatively in this series of dogs is 4.5 per cent.

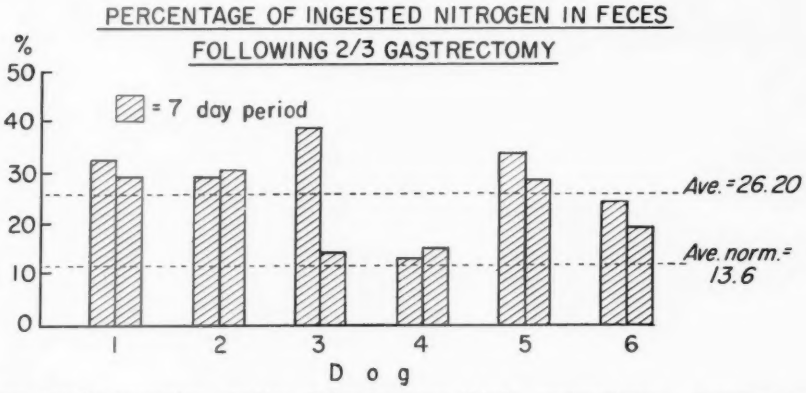


Fig. 2.—Nitrogen intake-excretion studies following two-thirds gastrectomy. Average control percentage of ingested nitrogen in feces in these six dogs is 14.9 per cent.

RESULTS

Partial Gastrectomy.—Under intraperitoneal Nembutal anesthesia a partial gastrectomy was performed on six dogs. Two-thirds of the stomach was removed in each case by accurate measurements of the lesser and greater curvatures. Polya's technique was used in each dog and the duodenal stump closed in the usual manner. After complete recovery from the operation, metabolic studies were repeated for two consecutive seven-day periods.

The average percentage values of ingested fat and nitrogen excreted in the feces in the preoperative periods of the gastrectomy group are 4.5 per cent and 14.9 per cent, respectively. An analysis of the results after the operation reveals that the excretions of fat and nitrogen in the feces are increased (Figs. 1 and 2). Statistical evaluation of these data appears to be highly significant ($P>0.01$).

There are sound physiologic reasons why this operative procedure might be expected to disturb fat and nitrogen assimilation. Some of the factors are as follows: (1) inadequate mixing of food with biliary and pancreatic secretions, since the food does not pass through the duodenum; (2) rapid passage of food through the stomach and the small bowel, thereby limiting adequate digestion and absorption; and (3) decreased pancreatic secretion and biliary drainage, due to lack of stimulation of such flow, since the food does not come in contact with the duodenum.[4, 5, 10, 18, 23, 35]

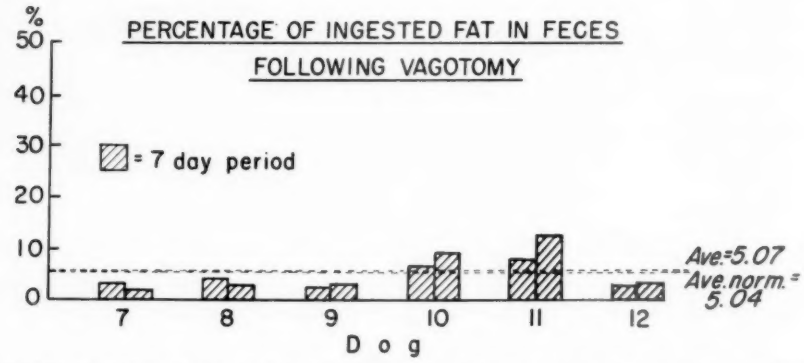


Fig. 3.—Intake-excretion studies of fat following vagotomy. Average normal percentage value of ingested fat in feces of these six dogs is 4.6 per cent.

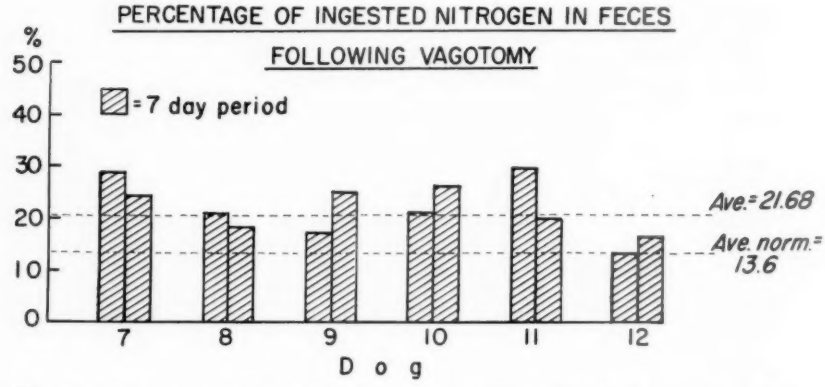


Fig. 4.—Nitrogen intake-excretion studies. The average control value in this series is 15.3 per cent.

These factors obviously disturb the necessary breakdown of food materials for proper absorption.

Vagotomy.—Supradiaphragmatic vagus section was performed on six dogs through a left thoracic approach. Branches of the vagus nerve were exposed in each case and sectioned carefully. The small fibers on the wall of the esophagus were also divided. Six weeks after surgery metabolic studies were repeated for two seven-day periods.

The average percentage values of ingested fat and nitrogen excreted in the feces in the preoperative periods of the vagotomy group were 4.6 per cent and 15.3 per cent, respectively. Statistical evaluation of the values after the opera-

tion indicates no noticeable change in the percentage of fat excreted in the feces (Fig. 3). Although nitrogen excretion appears to be somewhat elevated (Fig. 4), statistical analysis shows this change to be insignificant ($P>0.02$).

Gastroenterostomy With Vagotomy.—This operation has been recognized by many surgeons as a substitute for gastric resection in the treatment of peptic ulcer. Many favorable articles have appeared in the literature discussing the advantages of this combined procedure, but there appears to be no conclusive study comparing the nutritional effects of these procedures. With this goal in mind, a gastroenterostomy with vagotomy was performed on six dogs. In order to produce a uniform obstruction of the pyloric region, as in the case of patients with obstructed duodenal ulcer, the pyloric region was transected and the two

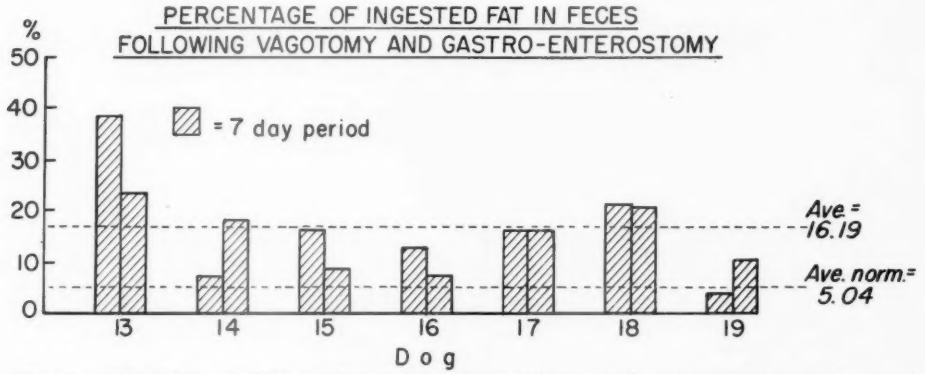


Fig. 5.—Metabolic studies of fat following vagotomy and gastroenterostomy. Preoperative average percentage value of ingested fat in feces of these seven dogs was found to be 5.9 per cent.

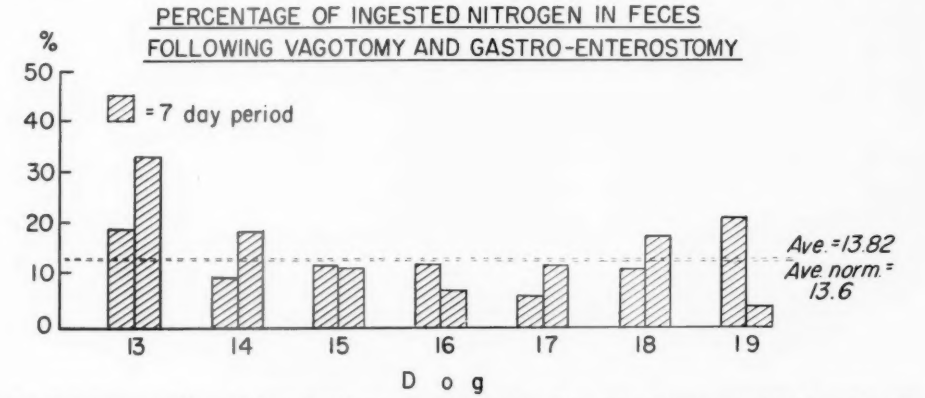


Fig. 6.—Intake-excretion determinations of nitrogen after vagotomy combined with gastroenterostomy. Average control percentage value of these dogs is 14.2 per cent.

cut ends closed individually. A posterior gastrojejunostomy was then accomplished in the usual manner, using the first loop of jejunum and placing the stoma close to the pyloric end. Nitrogen and fat studies were conducted on these animals after six weeks.

The average percentage values of ingested fat and nitrogen excreted in the feces in the preoperative periods of this series are 5.9 per cent and 14.2 per cent. When these data are compared with the values obtained postoperatively, it becomes clear that vagotomy combined with gastroenterostomy increases the

amount of fat excreted in the feces to 16.19 per cent (Fig. 5). Statistically, this change is significant ($P>0.005$). Nitrogen excretion, however, is not changed appreciably (Fig. 6).

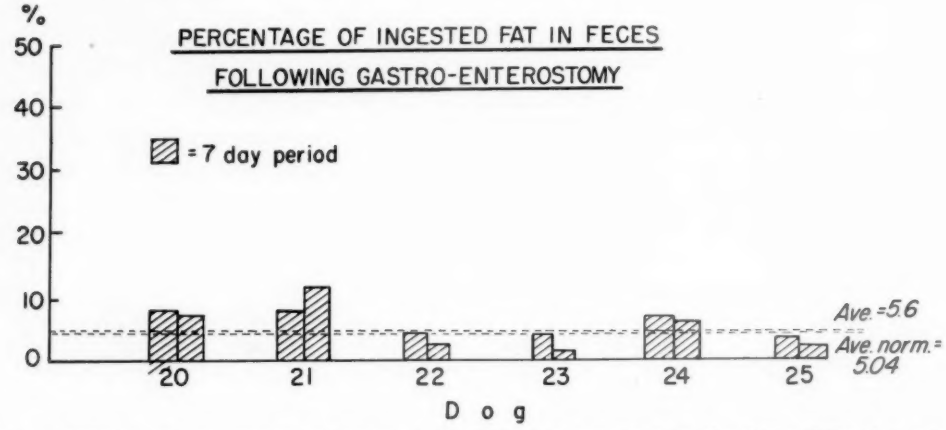


Fig. 7.—Studies on percentage of ingested fat which is excreted in the feces following gastroenterostomy. Average normal in this series is 4.5 per cent.

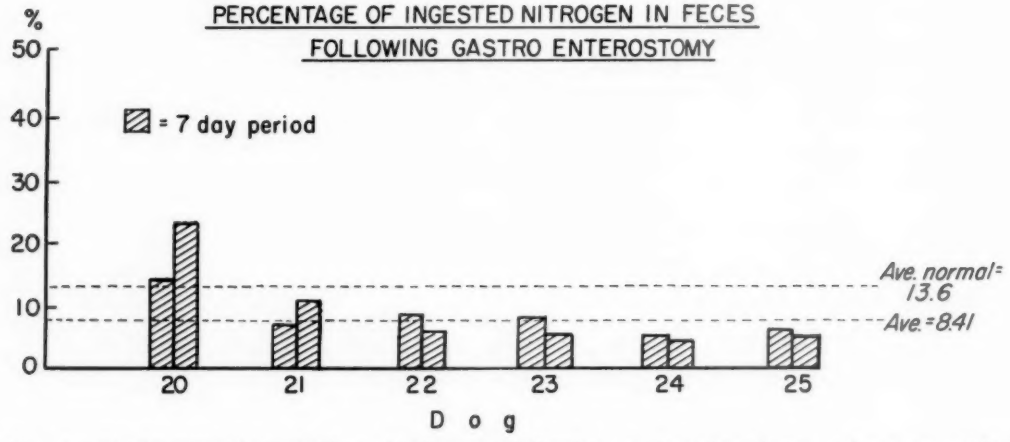


Fig. 8.—Intake-excretion studies of nitrogen following gastroenterostomy. Average control value is 9.9 per cent.

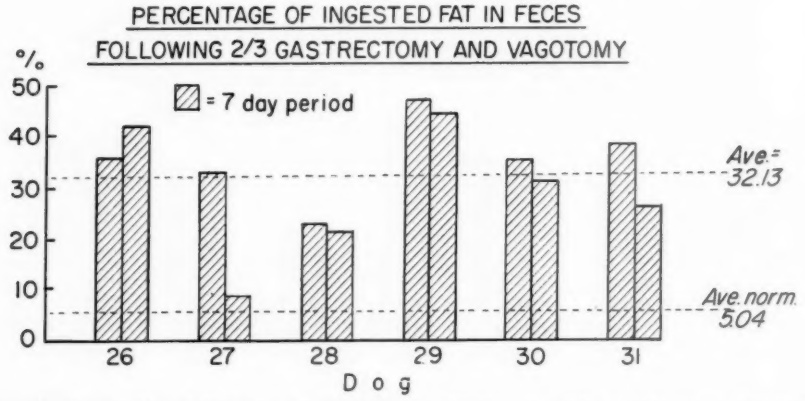


Fig. 9.—Metabolic studies following two-thirds gastrectomy and vagotomy. Average percentage value of the preoperative periods in this series of dogs is 5.7 per cent.

Gastroenterostomy.—This procedure is rarely used without vagotomy in the treatment of peptic ulcer, since the incidence of recurrence has been reported to be high. It is interesting, however, to study the effect of this procedure on fat and nitrogen assimilation. Six dogs were studied after gastroenterostomy (Figs. 7 and 8). An analysis of the results following this procedure revealed no significant change in fat and nitrogen assimilation.

Gastrectomy and Vagotomy.—According to our previous study the addition of vagotomy to gastric resection should increase the nitrogen and fat loss in the stool and therefore would be harmful to nutrition. In order to prove this point, vagotomy was combined with a two-thirds gastrectomy on six dogs, through an abdominal approach. Preoperative and postoperative metabolic studies were conducted as outlined previously. Figs. 9 and 10 clearly demonstrate that the percentages of ingested fat and nitrogen in feces following gastrectomy and

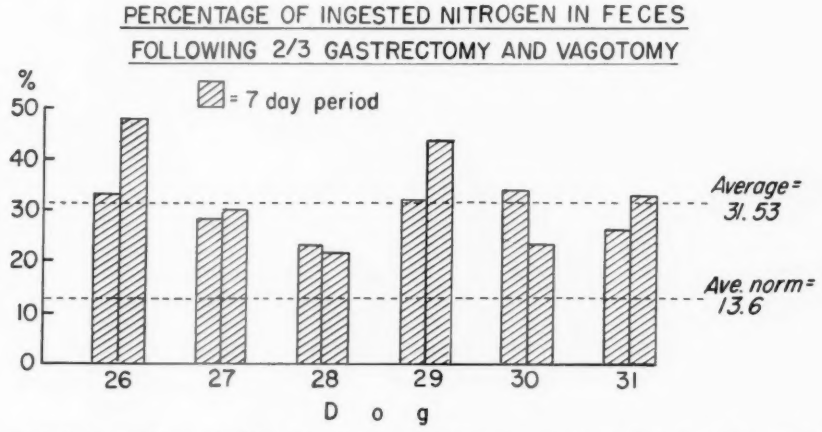


Fig. 10.—Intake-excretion studies of nitrogen. Average control value in these six dogs is 13.2 per cent.

vagotomy are considerably higher than normal. Statistical analysis of these data reveals significant change ($P>0.01$). When these values are compared with that in other gastric procedures, it appears that gastrectomy combined with vagotomy has the most harmful effect upon the nutrition.

SUMMARY

Metabolic studies were conducted on five series of dogs subjected to vagotomy, partial gastrectomy, gastroenterostomy, partial gastrectomy combined with cagotomy, and gastroenterostomy with vagotomy. Fat and nitrogen intake-excretion studies revealed that gastroenterostomy had no effect upon the percentage of fat and nitrogen output in feces. The combination of vagotomy and partial gastrectomy increased the fecal fat and nitrogen output to a degree considerably higher than gastrectomy alone. According to our findings it appears that gastroenterostomy combined with vagotomy is nutritionally a less harmful procedure than gastrectomy. Nitrogen excretion is increased to 26.2 per cent following two-thirds gastrectomy. A larger series is needed, however, to prove this point conclusively.

REFERENCES

1. Allen, A. W.: Discussion of paper by Lahey, F. H., and Marshall, S. H., Should Total Gastrectomy Be Employed in Early Carcinoma of the Stomach? Ann. Surg. 132: 540, 1950.
2. Allen, A. W., and Welch, C. E.: Gastric Ulcer, Ann. Surg. **114**: 498, 1941.
3. Anderson, Dorothy H., and Early, M. V.: Method of Assaying Trypsin Suitable for Routine Use in Diagnosis of Congenital Pancreatic Deficiency, Am. J. Dis. Child. 63: 891, 1942.
4. Bayliss, W. M., and Starling, E. H.: On the Uniformity of the Pancreatic Mechanism in Vertebrata, J. Physiol. **29**: 174, 1903.
5. Beal, John M., and Dineen, Peter: A Study of Vagotomy, Arch. Surg. **60**: 203, 1950.
6. Bell, Luther G., Sherer, Bernard, D., and Kennoy, John J.: Experiences With Subtotal Gastric Resection, Ann. Surg. **137**: 507, 1953.
7. Bohmansson, G.: Studien über die Chirurgische Behandlung von Gastroduodenal-geschwueren mit Besonderer Beruecksichtigung der Operation-sanastomie und der postoperativen Digestions Physiologie nebst einem Beitrag zur Frage der Chirurgischen Behandlung akuter Ulkus Blutungen, Acta chir. scandinav., supp. No. 7: 5, 1926.
8. Brain, R. H. F., and Stammers, F. A. R.: Sequelae of Radical Gastric Resections, Lancet **1**: 1137, 1951.
9. Browne, C. D., and McHardy, C.: The Patient After Gastric Surgery, South M. J. 36: 32, 1943.
10. Burns, H. H., and Holten, P.: The Standardization of Secretin and Pancreozymin, J. Physiol. **107**: 449, 1948.
11. Church, R. E., and Hinton, J. W.: Follow-up Results in Subtotal Gastric Resection for Ulcer, Am. J. Digest. Dis. **9**: 317, 1942.
12. Collins, E. N., Crile, G., Jr., and Dempsey, W. S.: Medical Follow-up of Vagotomy Plus Gastroenterostomy or Pyloroplasty for Peptic Ulcer, Ohio M. J. **46**: 33, 1950.
13. Colp, R., Klingenstein, P., Druckerman, L. J., and Weinstein, V. A.: A Comparative Study of Subtotal Gastrectomy With and Without Vagotomy, Ann. Surg. **128**: 470, 1948.
14. Crile, G., Jr., Jones, T. E., and Davis, J. B.: Surgical Treatment of Duodenal Ulcer. Comparison of Results With and Without Vagotomy, Ann. Surg. **130**: 31, 1949.
15. Dragstedt, L. R.: Discussion of Paper by Colp and co-workers.[13]
16. Dragstedt, L. R., and Woodward, E. R.: Appraisal of Vagotomy for Peptic Ulcer After 7 Years, J. A. M. A. **145**: 11, 1951.
17. Dragstedt, L. R.: Vagotomy in the Treatment of Peptic Ulcer, S. Clin. North America 32: 29, 1952.
18. Drewyer, C. E., and Ivy, A. C.: On Nonubiquitous Occurrence of Secretin, Proc. Soc. Exper. Biol. & Med. **27**: 186, 1929.
19. Edwards, L. E., and Harrington, Lynwood J., Jr.: Vagotomy and Gastroenterostomy—Vagotomy and Conservative Gastrectomy, Ann. Surg. **137**: 873, 1953.
20. Emery, E. S., Jr.: The Causes of Faulty Digestion in Dogs Without Stomachs, Am. J. Digest. Dis. **2**: 599, 1935.
21. Everson, T. C.: Nutrition Following Total Gastrectomy With Particular Reference to Fat and Protein Assimilation, Surg., Gynec. & Obst. **95**: 209, 1952.
22. Fox, H. J., and Grimson, K. S.: Defective Fat Absorption Following Vagotomy, J. Lab. & Clin. Med. **35**: 362, 1950.
23. Friedman, M. H., and Thomas, J. E.: The Assay and Distribution of Secretin, J. Lab. & Clin. Med. **35**: 366, 1950.
24. Gaviser, D.: Clinical Investigation and Evaluation of 416 Cases Consecutively Operated Upon for Peptic Ulcer, Proc. Staff Meet., Bull. Hosp. Univ. Minn. **19**: 81, 1947; SURGERY **24**: 873, 1948.
25. Gavrila, J., and Danicico, J.: Digestion des graisses et des protéines après résection de l'éstomac, Arch. mal. l'app. digest if. **19**: 587, 1929.
26. Gordon-Taylor, G., Hudson, H. V., Dodds, E. C., Warner, J. L., and Whitby, L. E. H.: The Remote Results of Gastrectomy, Brit. J. Surg **16**: 641, 1929.
27. Grimson, K. S., Rundles, R. W., Baylin, G. J., Taylor, H. M., and Linberg, E. J.: Vagotomy: Observations During Four Years, SURGERY **27**: 49, 1950.
28. Hedlund, E.: Quoted by Lublin.[34]
29. Hertel, Engelhard: Experimentelle Untersuchungen über den Einfluss der Magenresektion auf die Verdauung im Dünndarm, Arch. klin. Chir. **156**: 66, 1930.
30. Hoffman, W. S.: Photelometric Clinical Chemistry, New York, 1941, William Morrow & Co.
31. Ingelfinger, F. J.: The Late Effects of Total and Subtotal Gastrectomy, New England J. Med. **231**: 321, 1944.
32. Ivy, A. C.: The Effects of Gastrectomy in Animals, Am. J. Digest. Dis. **7**: 500, 1940.
33. Ivy, A. C., Grossman, M. I., and Bachrach, W. H.: Peptic Ulcer, Philadelphia, 1950, The Blakiston Company.

34. Lublin, R.: On Late Symptoms After Gastroenterostomy and Resection of Stomach (Billroth II) for Gastric and Duodenal Ulcer, Acta med. scandinav. Supp. No. 41: 1, 1931.
35. Mellanby, J.: Secretion of Pancreatic Juice, J. Physiol. **61**: 419, 1926.
36. Meyer, K. A., Brams, W. A., and Graves, W. N.: Final Results of the Operative Treatment of Gastric Ulcer, Arch. Surg. **81**: 1102, 1925.
37. Miller, E. R., and Oliver, J. H.: Results of Resection of the Vagus Nerves for Chronic Peptic Ulcer, Arch. Surg. **47**: 353, 1948.
38. Peters, J. P., and Van Slyke, D. D.: Quantitative Clinical Chemistry, Interpretations, Baltimore, 1946, Williams & Wilkins Company.
39. Ramson, H. K.: Subtotal Gastrectomy for Gastric Ulcer, Ann. Surg. **126**: 633, 1947.
40. Rekers, P. E., Abels, J. C., and Rhoads, C. P.: Metabolic Studies in Patient With Gastrointestinal Cancer; Fat Metabolism; Method of Study, J. Clin. Invest. **22**: 243, 1943.
41. Rekers, P. E., Pack, G. T., and Rhoads, C. P.: Metabolic Studies in Patients With Cancer of the Gastrointestinal Tract. VI. Disorders in Alimentary Digestion and Absorption in Patients Who Have Undergone Total Gastrectomy for Carcinoma of the Stomach, SURGERY **14**: 197, 1943.
42. Rekers, P. E., Pack, G. T., and Rhoads, C. P.: Metabolic Studies in Patients With Cancer of the Gastrointestinal Tract; Pancreatic Insufficiency in a Patient Treated Surgically for Carcinoma of Ampulla of Vater, J. A. M. A. **122**: 1243, 1943.
43. Shingleton, W. W., Fawcett, B., and Vetter, J. S.: Pancreatic Secretion and Response to Secretion After Vagotomy and Sympathectomy, Surg. Forum, Am. Clin. Coll. Surg., Philadelphia, 1951, W. B. Saunders Company, pp. 155-161.
44. Smith, F. H., and Jordan, S. M.: Gastric Ulcer, A Study of 600 Cases, Gastroenterology **11**: 575, 1948.
45. Snedecar, G. W.: Statistical Methods, ed. 4, Ames, Iowa, 1946, Iowa State College Press.
46. Strauss, A. A., Strauss, S., Levitsky, P., Scheman, L., Seidman, E. E., Arens, R. A., Myer, J., and Neckeles, H.: Physiological and Clinical Study of Patients After Subtotal Gastrectomy, Am. J. Digest. Dis. **4**: 32, 1937.
47. Tanturi, C. A., and Ivy, A. C.: On the Existence of Secretory Nerves in the Vagi for and Reflex Excitation and Inhibition of Bile Secretion, Am. J. Physiol. **121**: 270, 1938.
48. Von Haberer, H.: Ergebnisse nach weitgehender Resektion bzw. Totalexstirpation des Magens bei Karzinom und Ulkus, Deutsche. med. Wchnschr. **56**: 562, 1930.
49. Walters, W.: Total and Subtotal Gastrectomy for Carcinoma; Report of Case, Proc. Staff Meet., Mayo Clin. **12**: 465, 1937.
50. Walters, W.: Discussion on Dragstedt, Harper, Towe, and Woodward: Trans. Abdominal Gastric Vagotomy, Surg., Gynec. & Obst. **85**: 461, 1947.
51. Weaver, M. M.: Distribution of Pancreatic Secretion in the Gastrointestinal Tract, Am. J. Physiol. **82**: 106, 1927.
52. Welbourn, M. B., Hallenbeck, G. A., and Bollman, J. L.: Effect of Gastric Operations on Loss of Fecal Fat in the Dogs, Gastroenterology **23**: 441, 1953.
53. Wells, C., and Welbourne, R.: Postgastrectomy Syndromes; A Study in Applied Physiology, Brit. M. J. **1**: 546, 1951.
54. West, J. P.: Total Gastrectomy for Carcinoma of the Stomach, Ann. Surg. **129**: 373, 1949.
55. Wollaeger, E. E.: Disturbances of Gastrointestinal Function Following Partial Gastrectomy, Postgrad. Med. **8**: 251, 1950.
56. Wollaeger, E. E., Comfort, M. W., and Osterberg, A. E.: Total Solids, Fat and Nitrogen in the Feces. A Study of Normal Persons Taking a Test Diet Containing a Moderate Amount of Fat; Comparison With Results Obtained With Normal Persons Taking a Test Diet Containing a Large Amount of Fat, Gastroenterology **9**: 272, 1947.
57. Wollaeger, E. E., Comfort, M. W., Weir, J. F., and Peterberg, A. E.: The Total Solids, Fat and Nitrogen in the Feces: A Study of Normal Persons and of Patients With Duodenal Ulcer on a Test Diet Containing Large Amounts of Fat, Gastroenterology **6**: 83, 1946.
58. Wollaeger, E., Comfort, M. W., and Weir, J. F.: The Total Solids, Fat, and Nitrogen in the Feces: A Study of Persons Who Had Undergone Partial Gastrectomy With Anastomosis of the Entire Cut End of the Stomach and the Jejunum (Polya Anastomosis), Gastroenterology 6: 93, 1946.

MALIGNANT MELANOMA

Clinical and Pathologic Analysis of 93 Cases. Is Prophylactic Lymph Node Dissection Indicated?

Robert H. Lund, M.D., and Menard Ihnen, M.D., St. Louis, Mo.

(From the Department of Surgery and the Department of Pathology, Washington University School of Medicine, and the Barnard Free Skin and Cancer Hospital)

As early as 1902, Frederick Eve[1] emphasized that in addition to the excision of the primary lesion in the treatment of malignant melanoma, "the removal of the nearest chain of lymphatic glands, whether palpably enlarged or not, should never be omitted; for it may be taken as a matter of certainty that in the great majority of cases they are infected." Handley[2] (1907) showed by serial section that the circumferential permeation of the deep lymphatics takes place about the involved nodes as it does about the primary tumor. Therefore, simple excision of palpable regional nodes is not adequate, but wide "en bloc" excision is the treatment of choice. Ackerman and del Regato[3] (1947) stated that "when the tumor is located in an area from which the lymphatic drainage is predictable, a radical dissection of the anticipated metastatic node areas is mandatory." A similar position has been taken by most subsequent authors who have discussed the treatment of malignant melanoma. Curiously, there is no controlled data to support this thesis. Recorded series lack follow-up information[4] or there is a lack of correlation between clinical data, pathologic data, and follow-up.[5, 6]

It has been the practice at the Barnard Free Skin and Cancer Hospital to perform regional lymph node dissections only when the clinical impression indicated involvement by metastatic tumor (therapeutic dissections). It seemed worth while to study these cases in an attempt to obtain information about the value of prophylactic regional lymph node dissections in the treatment of malignant melanoma.

MATERIALS AND METHODS

Ninety-three cases of malignant melanoma* were treated and followed at the Barnard Free Skin and Cancer Hospital from May, 1935, through April, 1948.† Of 185 cases initially called malignant melanoma during this period, 65 cases were discarded because there was no record that the diagnosis had been microscopically confirmed by the institutional pathologist. Of the remaining 120 cases, all had either excisional or incisional biopsies prior to treatment. A histologic review revealed that 14 cases were junctional nevi in which a definitive diagnosis between melanoma and junctional nevus had not been made initially. Thirteen additional cases had been incorrectly diagnosed at the time of biopsy. The cases mistaken histologically

Received for publication Jan. 31, 1955.

*We are indebted to Dr. L. V. Ackerman for assistance in the review of some of the histologic sections.

†Melanomas originating in the eye, oronasal mucosa, and gastrointestinal tract were excluded. The term melanoma is used to signify those malignant tumors variously called nevocarcinomas, melanotic sarcomas, melanosarcomas, or melanocarcinomas and does not include moles, compound nevi, junctional nevi, or blue nevi.

for malignant melanoma were four junctional nevi, five pigmented subepidermal nodular fibroses, three blue nevi, and one sweat-gland carcinoma. Of the remaining 93 cases, four were lost to follow-up and 16 died from other causes within the five-year period. The remaining 73 cases serve as the basis of this study.

All of the patients in this group were indigent. They were predominantly farmers, laborers, and housewives from the St. Louis area and the surrounding rural areas of Missouri, Illinois, and Arkansas. All were Caucasians.

RESULTS

The sex distribution and five-year survival* rates are recorded in Table I. Survival rates for men and women are the same.

TABLE I. THE INCIDENCE AND FIVE-YEAR SURVIVAL OF MALIGNANT MELANOMA ACCORDING TO SEX

SEX	NO. OF CASES	FIVE-YEAR SURVIVAL	
		NUMBER	PER CENT
Male	38	8	21.1
Female	35	11	31.4
Total	73	19	26.0

Chi square of men versus women, $X^2 = 1.02$, d.f. $= 1$, $P = 0.30$. Conclusion: The five-year survival for the men and the women is the same.

Table II contains the survival rates by years after initial diagnosis at this hospital. Of the determinate cases, 26 per cent (19 of 73) survived for five years from initial treatment. By the seventh year, 21 9/10 per cent (16 of 73) had survived.

TABLE II. THE SURVIVAL RATE FOR 73 CASES OF MELANOMA

YEAR	1	2	3	4	5	6	7
No. of cases surviving	53	32	30	25	19	19	16
Per cent	76.6	43.8	41.1	34.2	26.0	26.0	21.9

Three of the 19 survivors died of generalized metastasis in the sixth year; one is living with recurrence noted at six and one-half years. Ten are alive without evidence of tumor and 5 died of causes unrelated to their melanoma.

The age distribution is shown in Table III. The ages varied from 19 to 89 years. The highest incidence was in the seventh decade. There were no prepubertal or pubertal melanomas in this group.

A history of definite antecedent trauma, such as cuts from shaving, irritation from clothing and shoes, bruises, and so on, was obtained in only seven instances. In an additional 11 patients there was trauma in the form of inadequate excision, electrodissection, irradiation, or application of caustic agents prior to their first visit to the Barnard Clinic.

In 62 cases, the melanoma was said to have been preceded by a nevus; no pre-existing nevus was present in four cases and in the remaining 27 no statement regarding pre-existence was recorded. None of these melanomas originated in hairy moles nor did any grossly resemble hairy moles. The nevi varied in color from light brown to jet black, ranging through shades of brown and blue. These nevi were flat, smooth, and usually less than 0.5 cm. in diameter.

*By five-year survival is meant a five-year postoperative period without evident recurrence; obviously, it does not indicate absolute cure. The validity of the five-year postoperative period is much greater with melanoma than with many malignant tumors. It has been shown that many patients with melanoma succumb early in the course.[7, 8, 9] An appreciable number of patients may also die after long intervals.[9, 10]

TABLE III. THE INCIDENCE AND FIVE-YEAR SURVIVAL OF MELANOMA ACCORDING TO AGE

AGE (YR.)	NO. OF CASES	FIVE-YEAR SURVIVAL		CHI SQUARE
		NUMBER	PER CENT	
0 to 10	0			
10 to 20	1	0	00.0	
20 to 30	5	2	40.0	
30 to 40	13	4	30.8	0.171
40 to 50	12	3	25.0	0 003
50 to 60	11	2	18.2	0.330
60 to 70	20	5	25.0	0.006
70 to 80	10	3	33.3	0.094
80 to 90	1	0	00.0	
				0.604

The interaction of heterogeneity chi square for this series using Brandt's formula

$$X^2 = \sum \frac{[(a_1 \times a_2t) - (a_2 \times a_1t)]^2}{a_1t \times a_2t \times n}$$

is 0.604, d.f. = 4, P = 0.95. Conclusion: The differences in the five-year survival according to age do not differ significantly from each other at the 5 per cent level (30 to 80 years).

TABLE IV. THE INCIDENCE OF PRE-EXISTING NEVUS

PRE-EXISTING NEVUS	NUMBER
Yes	62
No	4
Unknown	27
	93

The frequency of pigmentation and bleeding is recorded in Table V. The data available were inadequate to differentiate between traumatic and non-traumatic bleeding. The color of the melanomas varied from reddish brown to jet black and their form from flat to pedunculated.

TABLE V. THE INCIDENCE OF PIGMENTATION AND BLEEDING IN MELANOMA

	PIGMENTATION	BLEEDING
Yes	81	44
No	6	30
Unknown	6	19

The relationship between longevity and the size of the lesion is shown in Table VI. It is apparent that lesions less than 2 cm. in diameter had a more favorable prognosis. The survival in terms of duration of the lesion is shown in Table VII. No significant difference was found between the average size of the primary lesion in men and women.

TABLE VI. RELATIONSHIP OF SIZE OF LESION TO FIVE-YEAR SURVIVAL

SERIES	SIZE	NO. OF CASES	FIVE-YEAR SURVIVAL		CHI SQUARE
			NUMBER	PER CENT	
I	0 to 2 cm.	20	12	60.0	6.756
II	2 to 3 cm.	18	4	22.2	0.902
III	3 to 7 cm.	17	2	11.8	3.393
					11.051

Heterogeneity chi-square is 11.051, d.f. = 2, P = 0.01. Chi square testing the hypothesis that the mortality of Series I is significantly less than Series II and III.

	X^2	d.f.	P
I versus II	5.5464	1	0.02
I versus III	9.0898	1	0.001

TABLE VII. THE FIVE-YEAR SURVIVAL IN THE TREATMENT OF MELANOMA ACCORDING TO THE DURATION OF THE LESION PRIOR TO TREATMENT

SERIES	DURATION	NO. OF CASES	FIVE-YEAR SURVIVAL		CHI SQUARE
			NUMBER	PER CENT	
I	1 to 6 mo.	30	11	36.6	1.155
II	6 mo. to 2 yr.	24	6	25.0	0.098
III	2 to 5 yr.	7	0	00.0	2.704
					3.957

Heterogeneity chi square is 3.957, d.f. = 2, P = 0.10. Chi square testing the hypothesis that the mortality of Series I is significantly less than II and III.

	X^2	d.f.	P
I versus II	0.842	1	0.40
I versus III	3.652	1	0.05
II versus III	3.616	1	0.05

The distributional frequency and the survival rate relative to the location of the melanomas are shown in Table VIII. There were only four cases of subungual melanomas, including lesions of the toes in three instances and one case involving the index finger. The sole of the foot was involved in only one instance and there were no cases of melanoma involving the palm. These cases are included under the appropriate extremity.

TABLE VIII. THE FIVE-YEAR SURVIVAL IN THE TREATMENT OF MELANOMA ACCORDING TO LOCATION

LOCATION	NO. OF CASES	FIVE-YEAR SURVIVAL		CHI SQUARE
		NUMBER	PER CENT	
Head and neck	22	9	40.9	2.387
Trunk	21	4	19.0	0.582
Upper extremity	7	3	42.9	0.977
Lower extremity	21	3	13.3	1.929
				5.875

Heterogeneity chi square is 5.875, d.f. = 3, P = 0.20.

Twenty-nine patients (almost one-third) of the total group of 93 cases were inoperable when first seen because of widespread metastases. Therefore, only 64 cases were potential candidates for an attempt at surgical cure; all were treated by wide local excision, with or without lymph node dissections. As stated previously, regional lymph node dissections were performed only when it was believed clinically that the regional lymph nodes were involved. The number of survivors as related to the type of treatment is shown in Table IX. The five-year survival rate with wide local excision alone was 38.9 per cent. Among 27 individuals with proved regional nodal metastases who had therapeutic regional node dissections performed upon them, the five-year survival rate was 14.8 per cent.

The relationship between the clinical impression of the presence of lymphatic metastasis and survival is shown in Table X. Lymph nodes were considered involved in 32 cases. Nineteen of them had regional lymph node dissections performed and only one lived more than five years. The lone survivor did not have metastatic tumor in the excised lymph nodes. Dissection of the regional nodes was not performed on 13 and not one lived five years. The lymph nodes were felt to be uninvolved in thirty-eight cases. There were 13 patients in this group who survived for five years without evidence of metastasis or recurrence

following only wide local excision. Nine patients developed evidence of metastasis to the regional lymph nodes after the initial excision; regional lymph node dissections were performed upon these nine, and four patients survived for a five-year period. In all instances, there was microscopic evidence of lymph node metastasis. Considering the 38 patients on whom prophylactic node dissections might have been performed, 17 patients survived for five years. The relationship between the five-year survival, the lymph node area dissected, and the time of dissection are shown in Table XI.

TABLE IX. THE FIVE-YEAR SURVIVAL IN THE TREATMENT OF MELANOMA ACCORDING TO THE SURGICAL PROCEDURE

SERIES	OPERATION	NO. OF CASES	FIVE-YEAR SURVIVAL	
			NUMBER	PER CENT
I	Wide local cautery excision	7	1	14.3
II	Wide local scalpel excision	27	12	44.4
III	Wide local excision with regional node dissection	10	1	10.0
IV	Wide local excision with delayed regional node dissection	16	4	25.0
V	Amputation	2	1	50.0
VI	Amputation with regional lymph node dissection	2	0	00.0
		64	19	33.7

Chi square for Series I versus II is 2.140, d.f. = 1, P = 0.10; chi square for Series III versus IV is 0.891, d.f. = 1, P = 0.80.

TABLE X. THE FIVE-YEAR SURVIVAL IN THE TREATMENT OF MELANOMA ACCORDING TO THE CLINICAL IMPRESSION OF THE REGIONAL NODES

CLINICAL IMPRESSION	NO. OF CASES	FIVE-YEAR SURVIVAL	
		NUMBER	PER CENT
Involved	32	1	3.1
Uninvolved	38	17	44.7

Chi square for the involved versus uninvolved is 16.05, d.f. = 1, P = 0.001.

TABLE XI. THE FIVE-YEAR SURVIVAL OF MELANOMA ACCORDING TO THE REGIONAL AREA DISSECTED AND THE TIME OF DISSECTION

LOCATION	PRIMARY DISSECTION	FIVE-YEAR SURVIVAL		DELAYED DISSECTION	FIVE-YEAR SURVIVAL	
		NO.	PER CENT		NO.	PER CENT
Inguinal	6	0	00.0	7	3	42.9
Axillary	5	1	20.0	5	0	00.0
Neck	1	0	00.0	4	1	25.0

The primary lesion was available for histologic study in 64 cases. The depth of invasion in the available sections was arbitrarily classified into: (1) those limited to the very superficial dermis; (2) those within the remaining

TABLE XII. THE FIVE-YEAR SURVIVAL OF MELANOMA ACCORDING TO THE DEPTH OF INVASION OF THE PRIMARY TUMOR

SERIES	DEPTH OF INVASION	NO. OF CASES	FIVE-YEAR SURVIVAL	CHI SQUARE
I	Superficial	4	3	4.811
II	Dermal	42	12	0.086
III	Subcutaneous	18	2	2.203
				7.100

Heterogeneity chi square is 7.100, d.f. = 2, P = 0.02. Chi square for Series I versus II, 3.426, d.f. = 1, P = 0.05; chi square for Series I versus III, 7.606, d.f. = 1, P = 0.01.

dermis; (3) those which invaded the subcutaneous region. The results are recorded in Table XII. There was no relationship between the degree of pleomorphism and the prognosis.

DISCUSSION

The total survival rate of 26 per cent in this group is quite similar to those reported from other institutions.[6, 7, 10, 15, 17] A notable exception is a recent report by Meyer and Gumpert[16] in which the five-year survival rate in a group of 65 cases was 41.5 per cent. However, this type of comparison may have little meaning. Without exception, information is lacking in these reports which is necessary for a valid comparison of series. For example, the size of the lesions is probably important as a rough index of the duration of the tumor. Tumors larger than 2 cm. were associated with much lower survival rates in this series. It was observed that rural patients in this area allow visible lesions to progress to greater size than urban patients. One-fourth of the patients in this series had primary lesions less than 2 cm. in diameter, whereas one-half of those in a New York series had lesions of this size[9]; the proportion of cases having lesions of this type has rarely been available in reported series. The location of the primary lesion with respect to the regional lymph nodes is very important in evaluating the value of lymph node dissections. It is apparent that a lesion of the mid-abdomen or mid-back is unfavorably located for regional lymph node dissection, whereas axillary, upper pectoral, or inguinal lesions would be much more favorably located. Likewise, the degree of metastatic involvement of excised lymph nodes has been considered important in the prognosis of other tumors, such as carcinomas of the breast or colon; it probably has similar importance in malignant melanoma, especially if there is minimal or advanced metastatic involvement. This information is rarely given and is also unavailable in this study. In discussing the comparison of various series and survival data, Allen and Spitz[9] stated, "It is questionable if comparison of over-all results of two procedures performed by equally competent surgeons at different institutions are entirely equivalent. The comparison would seem by definition to require that similar lesions—similar in size, in depth of infiltration and in histological grade—lesions from comparable patients with regard to sex, age, and color—be used in the judgement of the respective merits of the procedures."

Turning to an analysis of the cases in this series, several findings are of interest, although the number of cases involved is small. It is discouraging that almost one-third of the total cases were beyond hope of cure when first seen because of widespread metastases. Education of patients and physicians may be of great benefit in reducing the number of these cases. A clinical impression of metastases to regional lymph nodes was almost always correct and was associated with an extremely poor prognosis. Regional lymph node dissection had no apparent value in this group. Therefore, regional lymph node dissections might have been of value only in the 38 cases in which the lymph nodes were not involved clinically (by definition, prophylactic dissections would be limited to this group). Since 13 patients in this group survived without removal of the regional

nodes, and regional nodes were removed in nine cases, improvement was theoretically possible only in the remaining 16 cases. This represents almost one-fifth of the total cases and nearly one-half of the group with clinically uninvolved nodes. The potential benefits would seem to warrant serious consideration of lymph node dissections. The results of treatment in the series recently reported by Meyer and Gumpert[16] are interesting. Five-year survival data were available on 40 patients on whom lymph node dissections had been performed. The group survival rate for five years was 52.5 per cent. Positive lymph nodes were present in 21 cases and seven survived; in 19 patients the lymph nodes were uninvolved and 14 of them lived five years (Chi square = 6.51, d.f. = 1, P = 0.01). The preoperative impression of the lymph nodes is not stated, nor is the extent of lymph node metastases given.

The treatment of choice of the primary lesion in malignant melanoma is wide, sharp excision. Electrocautery may be used if a very wide excision is performed so that the thermal necrosis does not extend to the tumor, rendering the tissue unsatisfactory for histologic examination for adequacy. It has been shown that compromise by inadequate sharp excision, or removal by freezing, electroneedles, or caustic agents, seriously jeopardizes the prognosis.[11-14] In our series there were 11 persons upon whom an inadequate excision of the proved lesion was performed prior to their first visit to this hospital. All of them developed local signs of persistent tumor and generalized metastasis.

Adair[12] found that only 2.5 per cent of melanomas treated by irradiation showed any response. None of the locally persistent primary lesions in this series treated by irradiation showed any response. Irradiation therapy has no place in the treatment of operable melanoma due to the high radioresistance of this tumor.

The place of prophylactic lymph node dissection in the treatment of melanoma is still controversial and unanswered by analysis of this series. It may be said that delay in dissection was not very successful and there is suggestive evidence that earlier lymph node dissections might have been beneficial. On the basis of the available evidence, considering the accepted principles in the treatment of many other tumors, and in view of the relatively low mortality and morbidity attendant upon lymph node dissections, it is our personal opinion that prophylactic dissections are indicated if the regional lymph nodes are in predictable and accessible areas.

SUMMARY

This study is based upon the experience gained from caring for 93 individuals bearing malignant melanomas of the skin, between 1935 and 1948.

Factors found to influence the prognosis in this series were:

1. *The size of the lesion:* Tumors smaller than 2 cm. in diameter were associated with greater longevity than those greater than 2 cm. in diameter.

2. *The palpability of regional lymph nodes:* A clinical impression of metastases to the regional lymph nodes was almost always correct and was associated with an extremely poor prognosis.

3. *The depth of invasion of dermis:* Tumors limited to the very superficial dermis were associated with much higher survival rates.

4. *The location of the melanoma:* Melanomas of the head and neck and upper extremities were associated with higher survival rates than those orginating on the trunk and lower extremities.

Factors previously deemed to have prognostic significance that were found to have none in this series are sex, age, and degree of pleomorphism.

The five-year survival rate following wide local excision alone was 38.9 per cent. In this series, 27 patients having proved nodal metastasis had therapeutic regional node dissections performed upon them. The five-year survival rate was 14.8 per cent.

The efficacy of the prophylactic removal of the lymph nodes in the treatment of malignant melanoma is discussed. Although the problem is unsettled, there is suggestive evidence that prophylactic lymph node dissection may be of value.

We are indebted to Dr. Richard E. Johnson for his statistical advice and comments.

REFERENCES

1. Eve, Frederick, A.: A Lecture on Melanoma, Practitioner **70**: 165-174, 1903.
2. Handley, W. S.: The Hunterian Lecture. The Pathology of Melanotic Growths in Relation to Their Operative Treatment, Lancet **1**: 927-933, 996-1003, 1907.
3. Ackerman, L. V., and del Regato, J. A.: Cancer; Diagnosis, Treatment and Prognosis. St. Louis, 1947, The C. V. Mosby Company.
4. Pack, G. T., Scharnagel, I., and Morfit, M.: The Principle of Excision and Dissection in Continuity for Primary and Metastatic Melanoma of the Skin, SURGERY **17**: 849-866, 1945.
5. Ackerman, L. V.: Malignant Melanoma of Skin. Clinical and Pathologic Analyses of Seventy-Five Cases, Am. J. Clin. Path. **18**: 602-624, 1948.
6. Daland, E. M., and Holmes, J. A.: Malignant Melanoma, A Clinical Study. New England J. Med. **220**: 651-659, 1939.
7. Nathanson, I. T., and Welch, C. E.: Life Expectancy and Incidence of Malignant Diseases, Am. J. Cancer **31**: 598-608, 1937.
8. Wright, C. J. E.: Prognosis in Cutaneous and Ocular Malignant Melanoma. A Study of 222 Cases, J. Path. & Bact. **61**: 507-525, 1949.
9. Allen, A. C., and Spitz, S.: Malignant Melanoma. A Clinico-pathological Analysis of the Criteria for Diagnosis and Prognosis, Cancer **6**: 1-45, 1953.
10. Ackerman, L. V.: Surgical Pathology, St. Louis, 1953, The C. V. Mosby Company.
11. Amadon, P. D.: Electrocoagulation of the Melanoma and Its Dangers, Surg., Gynec. & Obst. **56**: 943-946, 1933.
12. Adair, Frank E.: Treatment of Melanoma: Report of Four Hundred Cases, Surg., Gynec. & Obst. **62**: 400-409, 1936.
13. Pack, G. T., Perzik, S. L., and Scharnagel, I. M.: The Treatment of Malignant Melanoma: Report of Eight Hundred Sixty-Two Cases, California Med. **66**: 283-287, 1947.
14. Gage, M., and Dawson, W.: Malignant Melanoma, Ann. Surg. **133**: 772-781, 1951.
15. Pack, G. T., Gerber, D. M., and Scharnagel, I.: End Result in the Treatment of Malignant Melanoma, Ann. Surg. **136**: 905-911, 1952.
16. Meyer, H. W., Gumpert, S. L.: Malignant Melanoma: Appraisal of the Disease and Analysis of One Hundred Five Cases, Ann. Surg. **138**: 643-660, 1953.
17. Hall, J. R., Phillips, C., and White, R. R.: Melanoma. Study of 222 Cases, Surg., Gynec. & Obst. **95**: 184-190, 1952.

AN IMPROVED TECHNIQUE FOR AN ADEQUATE RADICAL GROIN DISSECTION FOR MALIGNANCY

STEPHEN L. GUMPORT, M.D., AND HERBERT WILLY MEYER, M.D.,
NEW YORK, N. Y.

(From the Tumor Service, Department of Surgery, New York University, Post-Graduate Medical School)

THE following technique for performing a radical groin dissection of both the superficial and deep inguinal and iliac lymph nodes has been developed by the Tumor Service of The Department of Surgery, New York University, Post-Graduate Medical School.

INDICATIONS

A radical groin dissection is indicated as a preventive or therapeutic procedure in the presence of a malignancy which may metastasize. It is performed if the primary lesion of the lower extremity, lower abdominal wall, buttock, external genitalia, perineum, and anus can be controlled, and if there are no signs of distant metastases. Lesions of the external genitalia, perineum, and anus require a bilateral radical groin dissection.

The greater number of our dissections have been performed for malignant melanoma, with squamous-cell carcinoma the second most frequent type of neoplasm. Occasionally an adenocarcinoma of the lower rectum may metastasize to the groin. One of our cases was performed for metastatic adenocarcinoma of the inguinal nodes secondary to a carcinoma of the sigmoid which had established a fistulous tract to the skin of the left lower quadrant of the abdomen. A radical groin dissection might also be utilized for a localized lymphoma.

Radical groin dissection is recommended for malignant melanoma whether or not lymph nodes are clinically palpable. In a previous study[4] we have reported microscopically involved nodes even in the absence of clinical evidence of involvement. With this fact in mind, especially with the seriousness of the prognosis, we advise adequate radical therapy for malignant melanoma. This is particularly true when we have an operation available, such as radical groin dissection, which can be performed with an excellent functional and cosmetic result and with a low morbidity and mortality rate.

Radical groin dissection is performed for malignant melanoma in the absence of clinically involved nodes several weeks after the primary site has been surgically controlled. This is done on the theoretical basis that melanoma cells may travel slowly to the regional nodes. If the node dissection is performed at the same time the primary lesion is removed, a shower of subcutaneous nodules may appear between the site of the primary tumor and the regional nodes.

Received for publication April 14, 1955.

TECHNIQUE

The night before operation the skin area is widely prepared, and the patient takes a tub bath. Fifteen hundred cubic centimeters of blood is typed and cross-matched.

On the operating table the patient lies on his back. The thigh on the affected side is slightly abducted and the leg externally rotated. The knee is slightly flexed over a sandbag. The scrotum is sutured to the opposite thigh.

The skin incision is made as shown in Fig. 1, *A*. It begins slightly cephalad to the anterior superior spine and about 2.5 cm. medial to it. It sweeps down in an ellipse to terminate in the mid-thigh over Hunter's canal.

Skin flaps are then raised with a thin layer of underlying fat over the anterior thigh, and with a slightly thicker layer of fat on the lower abdomen. Care should be taken not to extend the undermining of the flaps for more than 1.5 cm. beyond the margins of the landmark of the dissection.

Beginning on the abdomen the deeper fat and lymphatic tissues are dissected free from the underlying aponeurosis from above downward to the inguinal ligament. This aponeurosis is not removed with the specimen.

On the anterior thigh portion of the sharp dissection, not only is the fat and lymphatic tissue removed, but also the fascia of the sartorius, iliacus, pectineus, adductor longus, and rectus femoris muscles. This is performed dissecting alternatingly from the sides toward the middle and from below upward. The greater saphenous vein is divided both at the lower portion of the wound and at its junction with the femoral vein. The sheath of the femoral vein is removed.

The vascular and nerve supply of the sartorius muscle is carefully preserved as this muscle will be employed at the end of the procedure to cover the femoral vessels (Fig. 3, *C*).

The bulk of tissue making up the superficial groin dissection is dissected to a narrow neck attached on the medial side of the femoral vein at the femoral canal. This specimen is resected at this point. It is rarely necessary to resect a portion of Poupart's ligament unless nodes are firmly attached to it.

Fig. 1 depicts the operative field following the resection of the superficial node specimen.

The deep iliac node dissection follows. It is performed starting the incision as shown (broken line) in Fig. 1. This procedure is a modification of the method described by Cherney.[2, 3] The incision is made in the external oblique aponeurosis, opening the external ring, and is then extended across the rectus sheath to the midline. The underlying muscles are divided and the rectus is divided transversely down to the peritoneum. The peritoneum is bluntly dissected free beginning in the region of the space of Retzius and retracted cephalad. The deep epigastric vessels are first ligated and divided. At times, an anomalous obturator artery arises from the deep epigastric artery and may have to be divided. The external iliac vessels should be dissected free of all lymph nodes, fat, and areolar tissue up to a point where the hypogastric artery arises (the

ureter may be noted at this point) and even higher if necessary. The lateral margin of this deep dissection is the psoas muscle, and the medial margin is the obturator nerve. Care must be taken to remove the group of nodes lying just behind the ramus of the pubis close to the obturator foramen. Fig. 2 shows the completed deep and superficial dissection.

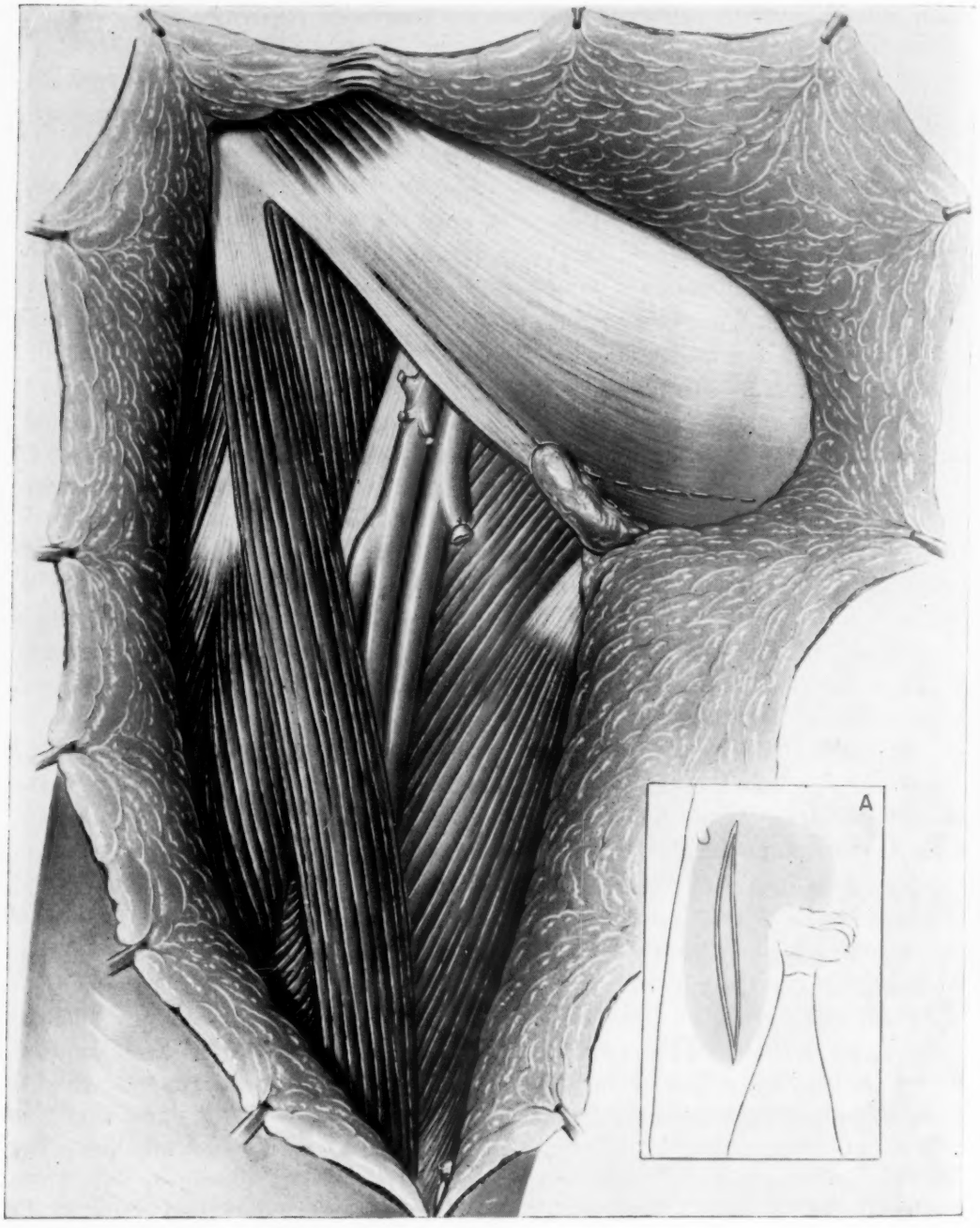


Fig. 1.—Completed femoral and inguinal node dissection field. Inset *A* shows average skin incision and shaded area is extent of dissection. The dashed lines indicate incision of aponeurosis of external oblique and rectus sheath.

The peritoneum and its contents are then allowed to fall back into their normal position. The superior divided end of rectus muscle is sutured to the lower flap of anterior rectus sheath.[3] Then the incision in the external oblique aponeurosis and the rectus sheath are sutured. Following this, the potential space on the medial side of the femoral vein is closed by suturing the inguinal ligament to Cooper's ligament and the pectineus muscle.

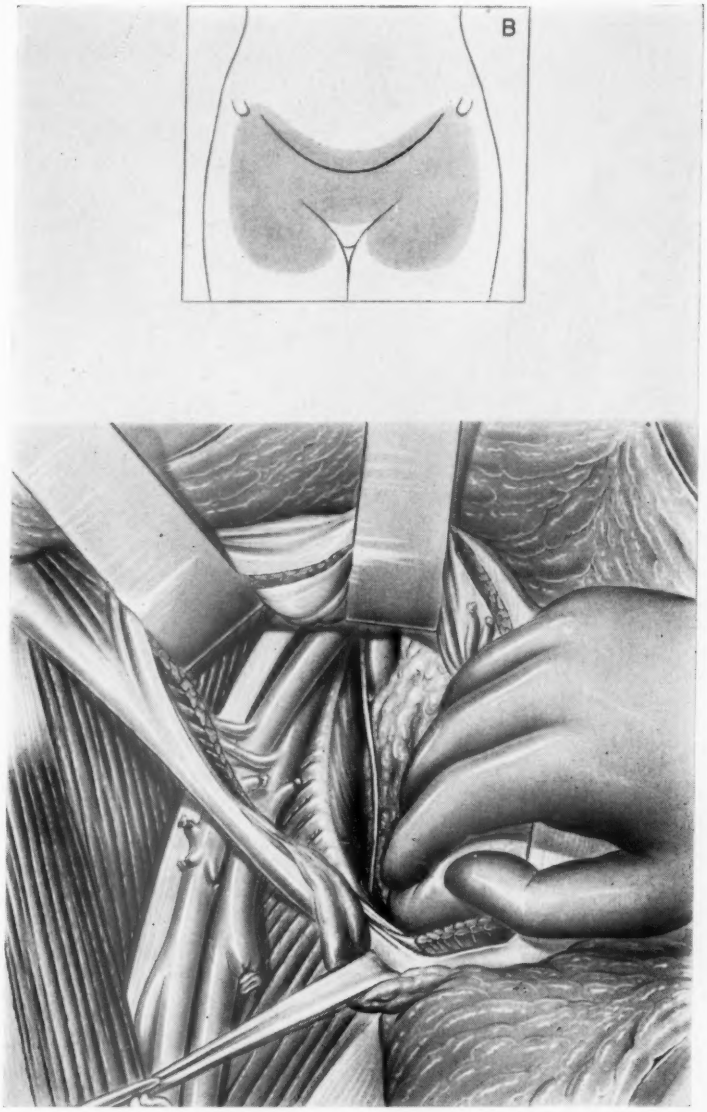


Fig. 2.—Completed deep iliac node dissection. Note that inguinal ligament is not divided but note excellent exposure after dividing external oblique and internal oblique muscles and lower rectus muscle transversely. Inset *B* shows skin incision for bilateral groin dissection and shaded area is extent of dissection.

The sartorius muscle is then isolated close to the anterior superior spine. It is divided transversely and swung or rotated medially to cover the femoral nerve and vessels.[1] The cut edge is sutured to Poupart's ligament and the lateral

borders to the iliacus, adductor longus, and pectineus muscles. In this way the femoral nerve and vessels are protected and the dead space over Scarpa's triangle is obliterated. Fig. 3 shows the operative field completed and ready for skin closure.

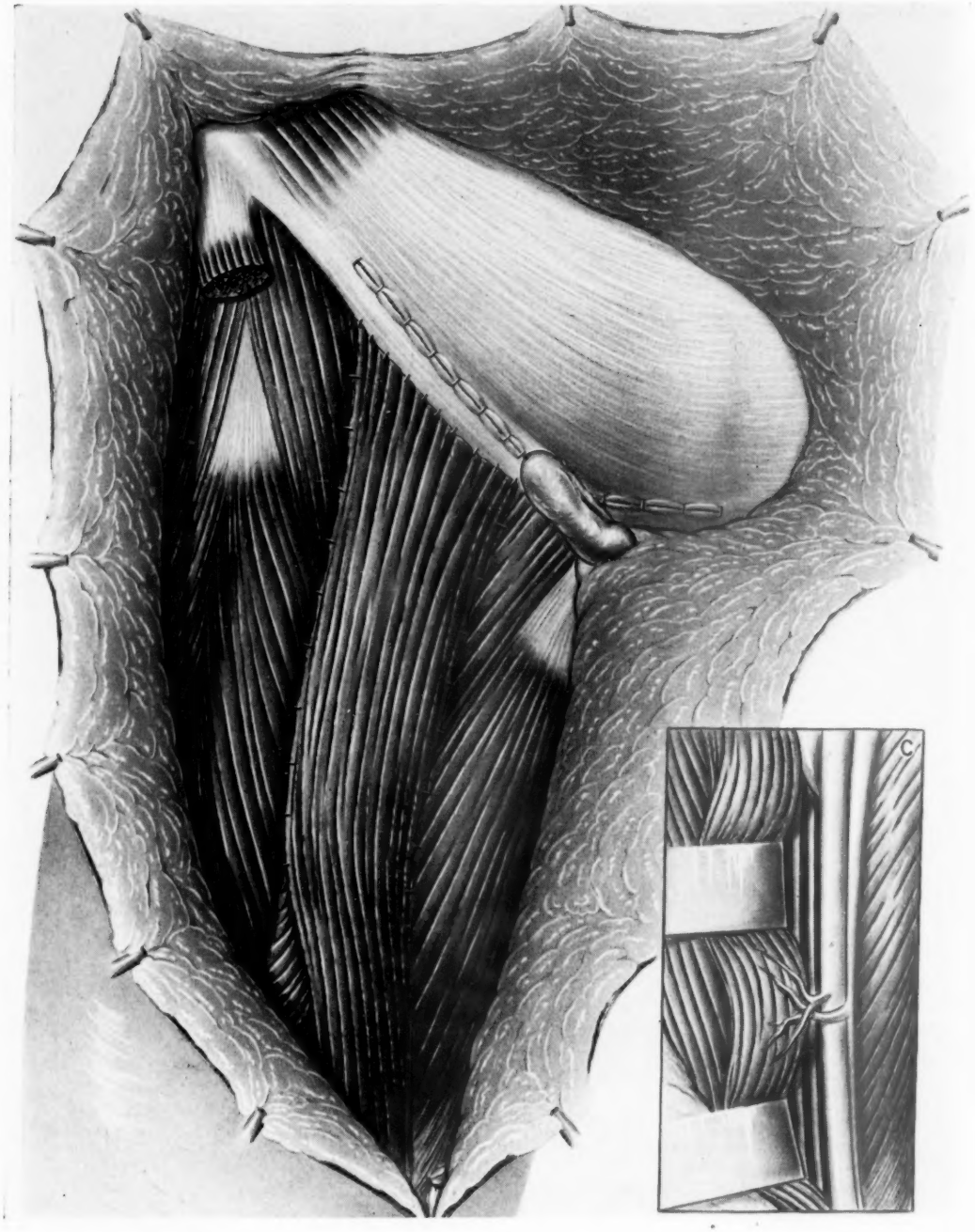


Fig. 3.—Completed field after suture and reconstruction of inguinal canal and rectus sheath. Note that femoral vessels and nerve are covered by sartorius muscle. Inset *C* shows preservation of blood supply to sartorius muscle.

Penrose drains may be employed, bringing them out through the superior and inferior angles of the incision or through an outer, lateral stab wound. Recently we have employed suction drainage through two-multi-eyed urethral catheters to the upper and lower portions of the wound. They are connected with a glass Y tube and hooked up to a Wangensteen suction or preferably an electric suction pump, if available.

Ordinarily, cotton or silk technique is used throughout. However, if the field is considered an infected one, catgut is employed.

Ace bandages are applied to the entire lower extremity. Suction is maintained for three to five days. Postoperatively, Ace bandages are worn for at least six months to minimize the lymphedema which may occur.

Patients are kept in bed for about ten days. The skin sutures are not removed before ten to fourteen days.

BILATERAL RADICAL GROIN DISSECTION

A great advantage of the procedure described here is that it may be extended to perform simultaneous bilateral node dissections when indicated. The procedure is essentially the same as that described for the unilateral dissection except that the skin incision is made as shown in Fig. 2, *B*. The superficial portion of the dissection is performed essentially as described previously. In certain lesions, other than of the lower extremity the dissection does not have to be carried as far inferiorly on the thighs. The deep iliac portion of the dissection is carried out by extending the incision, shown as a broken line in Fig. 1, across the midline and up the opposite side. This will give the necessary exposure for the performance of the deep dissection.

COMPLICATIONS

The more common postoperative complications are necrosis of the skin edges, separation of the suture line, lymphedema, and serum collection. Necrosis of the skin edges may be minimized by employing great care and good judgment in not undermining the skin flaps more than necessary. Separation of the skin edges may be prevented by bed rest for ten to fourteen days and leaving the skin sutures in place at least that long or longer. However, if either of these two complications occurs, a split-thickness graft placed on the underlying transplanted sartorius muscle will remedy it. Primary skin graft may be indicated if much skin must be sacrificed or if there is evidence of tension at time of skin suture. Lymphedema may be lessened by Ace bandages, frequent elevation of the extremity, and avoiding unduly prolonged standing.

DISCUSSION

Adequate radical surgery is indicated in the efficient treatment of malignancy. Excision of the primary lesion in continuity with the regional lymphatic drainage area is an important principle. When this procedure is not feasible for technical reasons, regional node dissections must be performed as separate procedures. An adequate radical groin dissection usually presents various hazards. The ilioinguinal ligament was formerly divided, and access to the deep

iliac nodes was frequently inadequate. An improved simple technique is described in which the ilioinguinal ligament is preserved. However, excellent access is obtained for the deep iliac node dissection by dividing either one or both recti muscles transversely just above their insertion into the pubis.

CONCLUSION

An apparently improved technique for radical groin dissection has been presented. It may be employed for unilateral or bilateral dissections. Indications for these procedures are briefly discussed.

The illustrations for this article were executed by Philip C. Johnson, Assistant Professor of Surgery (Medical Artist), Department of Surgery, New York University, Post-Graduate Medical School.

Appreciation is extended to Dr. Herbert Newman, Associate Clinical Professor of Surgery, New York University, Post-Graduate Medical School, for his helpful suggestions in regard to the development of the one-stage bilateral radical groin dissection.

REFERENCES

1. Baronofsky, I. D.: Technique of Inguinal Node Dissection, SURGERY 24: 555-567, 1948.
2. Cherney, L. S.: A Modified Transverse Incision for Low Abdominal Operations, Surg., Gynec. & Obst. 72: 92-95, 1941.
3. Cherney, L. S.: Transverse Low Abdominal Incision With Detachment of the Recti From the Pubis, J. A. M. A. 157: 23-26, 1955.
4. Meyer, H. W., and Gumport, S. L.: Malignant Melanoma, Ann. Surg. 138: 643-660, 1953.

THE USE OF A TRIPLE-LUMEN SINGLE-BALLOON TUBE IN THE DIAGNOSIS AND TREATMENT OF MASSIVE UPPER GASTROINTESTINAL HEMORRHAGE

MARVIN M. NACHLAS, M.D.,* BOSTON, MASS.

(From the First [Tufts] Surgical Service of the Boston City Hospital, and the Department of Surgery, Tufts College Medical School)

INTRODUCTION

FEW patients present a more difficult diagnostic and therapeutic challenge than the one who sustains a massive upper gastrointestinal hemorrhage. Great strides have been made in the handling of this catastrophe, in large part due to the emphasis on the diagnosis of the source of the hemorrhage.

Understandably, many physicians have been reluctant to expose a patient who is actively bleeding to the rigors of a roentgen examination. There has always been some doubt as to the accuracy of the roentgenologic procedure under these circumstances, and the fear is ever present that the trip to the fluoroscopy room and the stimulation of peristaltic activity by the ingested barium will lead to an exacerbation of the bleeding. Although these reservations have not been entirely dispelled, the detailed studies of Hampton,[1] Schiff,[2] Schatzki,[3] Jones,[4] Allen,[5] Ritvo and associates,[6] and others who advocated early x-ray examination have conclusively demonstrated that the assistance obtained from the roentgenologist in making the early diagnosis far outweighs the risk of the examination and obviates, in many cases, the need to attempt treatment without a diagnosis. The value of x-ray studies in the acute bleeder is well documented.[1, 3, 6, 7] Unfortunately, there are a significant number of patients who are either bordering on or in the shock state, who cannot be transported to the fluoroscopy table. These are often the patients who are in most desperate need of an early diagnosis. During the three-year period from 1948 to 1951, there was a group of men at Boston City Hospital specifically engaged in seeing these acute bleeders and trying to arrive at an early diagnosis.[6] Nevertheless, in only 74 of 123 patients (60 per cent) were they able to carry out the x-ray studies within the first forty-eight hours of admission.

More recently, the use of the sulfobromophthalein sodium (BSP) retention test as a screening tool for cirrhosis of the liver has proved valuable in alerting the diagnostician to the possibility of esophageal varices.[7] When the degree of retention is less than 5 per cent at the end of forty-five minutes, the likelihood of varices is remote, when between 5 per cent and 15 per cent the

Received for publication Feb. 22, 1955.

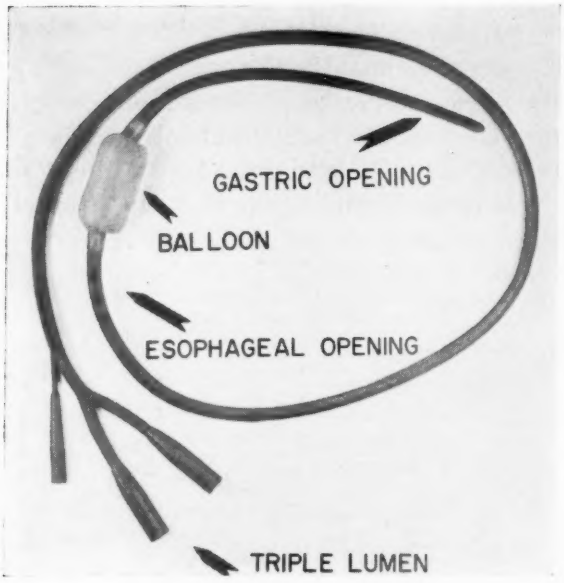
*Present address: Sinai Hospital of Baltimore, Inc., Baltimore 5, Md.

possibility exists, and when greater than 15 per cent the diagnosis of esophageal varices must be seriously entertained, and efforts to identify them should be made. Several limitations to the value of this test result from the fact that severe shock may lead to an elevation of the per cent of dye retained; that variceal bleeding from extrahepatic portal obstruction would be missed because of the adequate liver function in those cases; and that it is a common finding to have patients with liver disease who are bleeding from a source other than varices. For example, in a study by Chalmers and associates[8] of 101 patients dying of an upper gastrointestinal hemorrhage at the Boston City Hospital during the years 1945 to 1949, 44 were found to have cirrhosis of the liver, but only 32 bled from varices. Thus, there were 12 patients (12 per cent) who had liver disease but who bled to death from a cause other than esophageal varices! Despite these limitations, there is little doubt that the BSP retention test is a most helpful adjunct in the diagnosis of massive upper gastrointestinal hemorrhage.

Additional help in the localization of the bleeding point has been obtained by esophagoscopy. Despite the difficulties encountered in examining a desperately ill patient under topical anesthesia and at the bedside, it has usually been possible to identify the source of the bleeding.[9] Unfortunately, this reported enviable degree of accuracy is not universally attainable, nor is the procedure entirely free of risk. The lack of cooperation exhibited by some patients often makes esophagoscopy impractical.

Despite these important advances, massive bleeding continues to tax the clinical acumen of the physician charged with its management. Frequently, therapy is not entirely a matter of the exact diagnosis of the lesion, but whether the bleeding is occurring from above or below the diaphragm. This is especially true when surgical intervention is being considered. Various plans of management have been proposed for the patient hemorrhaging from his gastroduodenal area[2, 5, 7, 10, 11, 12] (most commonly due to peptic ulcer); and whichever one is adopted, the outcome may well be favorable as long as the bleeding point is located in the area which is being treated, either medically or surgically. In contrast, the handling of lesions above the diaphragm is usually governed by different principles, and a mistaken laparotomy for esophageal pathology, such as varices, may prove fatal. Reports attesting to the value of esophageal balloon tamponade have been in the literature since 1930.[13] Recently, modifications of the Miller-Abbott tube or the use of both esophageal and gastric balloons have effectively controlled bleeding from varices.[14-21]

This investigation of the diagnostic and therapeutic possibilities of a triple-lumen single-balloon tube in the patient with massive bleeding was stimulated by the two observations mentioned previously; namely, (1) that the bleeding site must be localized in relation to the diaphragm, and (2) that a single balloon occluding the cardioesophageal junction does control bleeding from the esophageal varix. It is the purpose of this paper to report our experience with the use of such a tube at the Boston City Hospital.



A.

B.

Fig. 1.—*A*, The single-balloon triple-lumen tube, demonstrating the openings for drainage of the esophagus and the stomach, which are connected to separate suction bottles.

B, One of the early tubes used shortly after removal from Case 7. Since the lumen draining the esophagus was too small, the blood did not drain completely and some clotted around the esophageal portion of the tube. There was no doubt as to the bleeding source in this patient.

RESULTS

During the past eighteen months the tube has been successfully passed* on 20 patients with severe upper gastrointestinal hemorrhage. In most instances, there were some unusual features about the cases that led to the use of the tube. Many of those who bled from peptic ulcer were handled in the usual fashion by the house staff and were not brought to our attention. Table I lists the impressions concerning the source of the hemorrhage which were

TABLE I. SOURCE OF HEMORRHAGE IN PATIENTS WITH MASSIVE UPPER GASTROINTESTINAL BLEEDING

CASE	CLINICAL IMPRESSION*		BSP RETENTION (PER CENT)	TUBE DIAGNOSIS	X-RAY FINDINGS	FINAL DIAGNOSIS†
	ESOPHAGUS	STOMACH				
1	3	0	30	Stomach	Gastric ulcer	Peptic ulcer (roentgen). Cirrhosis of the liver
2	3	0	78	Esophagus	Varices	Esophageal varices (operation)
3	0	3	0	Stomach	Too ill for examination	Duodenal ulcer (operation)
4	3	0	30	Esophagus	Normal G.I.	Esophageal varices (post-mortem)
5	2	1	42	Esophagus	Too ill	Esophageal varices (clinical)
6	1	0	--	Esophagus	Varices?	Esophageal varices (clinical)
7	3	0	33	Esophagus	Normal G.I.	Esophageal varices (operation)
8	1	1	28	Esophagus	Too ill	Esophageal varices (operation)
9	0	2	60	Esophagus	Normal G.I.	Esophageal varices (clinical)
10	0	2	18	Stomach	Too ill	Duodenal ulcer (operation)
11	3	0	23	Esophagus	Normal G.I.	Esophageal varices (clinical)
12	1	0	40	Esophagus	Varices?	Esophageal varices (P. M.)
13	1	1	22	Esophagus	Normal G.I.	Esophageal varices (P. M.)
14	0	3	15	Esophagus	Hiatus hernia Esophageal ulcer?	Hiatus hernia (roentgen)
15	1	1	45	Stomach	Esophageal varices Duodenal ulcer	Duodenal ulcer—cause of hemorrhage (roentgen)
16	0	3	3	Stomach	Gastritis	Gastritis (roentgen). Cirrhosis of liver
17	2	1	40	Esophagus	Esophageal varices Gastritis	Esophageal varices (operation)
18	1	2	47	Stomach and esophagus	Too ill	Duodenal ulcer (P. M.). Esophageal varices (P. M.)
19	0	1	40	Esophagus	Normal G.I.	Varices (esophagoscopy)
20	0	3	9	Stomach	Gastritis	Gastritis (roentgen)

*The clinical impressions as to the source of the hemorrhage were usually made after the history, physical examination, and the BSP retention studies were completed. Most patients were seen by three different physicians, and the numbers recorded in these columns indicate the number of physicians favoring each location for the source of the hemorrhage.

†The final diagnoses were verified by either operative, roentgenologic, esophagoscopic, post-mortem, or clinical means. This last designation refers to cases where tamponade was effective or the patient went into hepatic coma during or after the gastrointestinal hemorrhage.

obtained on purely clinical grounds, following the use of the tube, and following roentgenologic examination. The means by which the final diagnoses

*The tubes used in this investigation were manufactured by the Davol Rubber Company, Providence 2, Rhode Island. Details of the method of employing the tube and the interpretation of the findings during its use will be distributed with each tube.

were established is also recorded. Most patients who were bleeding on admission were examined by the admitting doctor, the intern on the ward, and the resident. In only 6 out of 20 cases did the diagnosis seem so definite that a single diagnosis was advanced by all of the house officers who saw the patients. In the other 14, when more than one diagnosis was listed, the first impression was recorded in the table; and in 6 of these cases there was disagreement between the examining physicians as to the source of the hemorrhage. It may be noted also that the Bromsulphalein retention test and x-ray examination were not always enlightening. The roentgen findings were of little help in 6 cases, and 5 other patients were too ill to be examined roentgenologically. All of these patients were bleeding massively and required from two to twenty-two transfusions to maintain a normotensive level. Most of the patients who received only a few transfusions died within a short time after admission. In most instances, the bleeding source was determined by the tube as quickly as possible and without our knowledge of the BSP test. Impressions based upon the use of the tube were later verified by either roentgen, operative esophagoscopic, or post-mortem examination in 18 cases, while in the 2 others the lesion could not be determined accurately. The cases whose final diagnoses rested on clinical grounds may be considered unproved, but, in reality, the tube findings were consistent with the subsequent course of the patient, following the indicated therapy.

In addition to these 20 patients, there were 4 in whom the tube could not be passed because of their poor cooperation or in whom the tube was passed after their hemorrhage had ceased and thus the source could not be determined. In 13 patients, tamponade was indicated and attempted, with apparent control of the hemorrhage in 10 and no effect in 3. In the latter 3 cases, the single-balloon tube was replaced by the double-balloon (Sengstaken-Blakemore)[21] for lateral esophageal compression, but the hemorrhage was still uncontrollable.

DISCUSSION

Many cases of upper gastrointestinal bleeding can be accurately diagnosed after an adequate history report and physical examination. Others require all the means at our disposal and often we are still uncertain as to the source. The incidence of the undiagnosed cases among the bleeding population of different hospitals varies from a low value of 2 per cent to a high of 26.4 per cent.[2, 23-26] This variation probably results from several factors, such as, (1) the type of bleeders admitted to the institution, (2) the criteria for a massive hemorrhage, (3) the laxity or strictness in accepting the findings of a small hiatus hernia, gastritis, duodenal deformity, diverticula, and duodenitis as a cause for the hemorrhage, and (4) the number of cases in each series examined at laparotomy or post-mortem examination. Stone,[27] Gray and co-workers,[28] and Crohn and associates[23] have published illuminating papers devoted to the problems of the undiagnosed bleeders. Because a large quantity of blood can be lost from an almost microscopic lesion, and because our diagnostic tools are in the main macroscopic, there will probably always be

some patients in whom the source of the bleeding cannot be identified. The drawbacks of the Bromsulphalein retention test have already been mentioned. Roentgen examination in the presence of active hemorrhage and a stomach filled with blood is always a difficult undertaking, and not infrequently the patients are too ill to be transported to the fluoroscopy room. Esophagoscopy is limited to those hospitals where well-trained endoscopists are readily available, and even in their hands this procedure may be quite trying when blood is constantly appearing in the field. Furthermore, when the bleeding has ceased, how often can the observed varices be incriminated as the source? Despite all of their shortcomings, the BSP retention test, early roentgen studies, and esophagoscopy have brought many previously obscure cases into the diagnosed group. The triple-lumen single-balloon tube is suggested as an additional diagnostic tool which involves little risk to the patient, a minimum of discomfort, and may identify the source of the hemorrhage in relation to the diaphragm in a relatively short time.

Besides its diagnostic usefulness, the triple-lumen single-balloon tube can effectively tamponade some patients bleeding from esophageal varices. Previous reports describing successful tamponade have included both single- and double-balloon tubes, and it has been asserted by the individuals who use the single balloon[16, 18, 29] that lateral esophageal compression is unnecessary. A review of the anatomy involved will help to clarify this viewpoint. Blood flowing through the esophageal varix comes from two sources, namely, (1) through their connection with the submucosal veins of the stomach which cross the cardioesophageal junction, and (2) from the periesophageal veins which send perforators through the muscular coats of the esophagus to anastomose with the submucosal varices. Since the periesophageal veins drain freely into the azygos veins which maintain a negative venous pressure, it seems likely that the greater portion of the blood in the periesophageal veins will continue flowing toward the heart rather than at right angles through the muscular wall of the esophagus. Some variation will occur from case to case depending upon how numerous and how well developed these communicators are. That the major flow comes from the submucosal gastric veins is evidenced by the fact that tamponade with a single balloon at the cardioesophageal junction is effective in many cases. When a significant portion of the flow is from the periesophageal veins through large communicating veins to the submucosal varices, then lateral esophageal compression may be of added value. In my experience, if obstruction of the venous drainage from the submucosal gastric veins does not control the hemorrhage, lateral pressure within the esophagus will also fail.

However, until further experience substantiates this viewpoint, it is recommended that when the single-balloon tube has identified the bleeding as esophageal in origin and tamponade is not satisfactory, it should be replaced by a Patton or Sengstaken double-balloon tube.

Two additional advantages accrue from the use of the triple-lumen single-balloon tube. First, observations upon the continuance of the bleeding and its severity are easily made by watching the drainage from the two lumens

emptying the stomach and the esophagus. Second, the tube may be left in place while the patient who requires surgery is being anesthetized, thus avoiding the hazard of aspiration from the blood-filled stomach.

There are several lesions which we have not as yet encountered, where, on theoretical grounds, the tube may prove valueless. A gastric ulcer high on the lesser curvature of the stomach, a malignancy in the cardia, or a gastric varix might be impossible to identify. However, it is entirely conceivable that under these circumstances the hemorrhage may be controlled by the tamponade and permit the other diagnostic procedures to be carried out under more favorable conditions. Some of the therapeutic possibilities of the tube still have not been explored. Can all hemorrhages from esophageal ulcers be controlled? Will trapping the blood in the stomach pouch of a hiatus hernia tamponade the bleeding? Does compression of part of the stomach significantly reduce the hemorrhage occurring from gastritis? The answers to these questions should come with further experience. In the meantime, the triple-lumen single-balloon tube is offered as an adjunct to be used with other established aids in the diagnosis and treatment of the patient who is massively bleeding from his upper gastrointestinal tract.

SUMMARY

1. A triple-lumen single-balloon tube has been described for diagnostic and therapeutic use in patients with massive upper gastrointestinal hemorrhage.

2. The tube has been used in 20 patients and the source of the hemorrhage determined in 18, as proved by operative, post-mortem, or roentgenologic findings. In the two other patients, the tube findings could not be verified by the usual clinical techniques, but the subsequent course of the patient was consistent with the impressions gained from the use of the tube.

3. Tamponade was utilized in the 13 patients with esophageal bleeding. In 10 of these patients, it was partly or completely satisfactory. In the 3 others, the bleeding was so active that neither the single-balloon nor the double-balloon tubes were effective.

4. Although further studies are indicated for a final evaluation, the use of the triple-lumen single-balloon tube has been of sufficient help to us to offer it as another diagnostic and therapeutic tool in the management of the patient who is bleeding actively from the upper gastrointestinal tract.

I would like to express my appreciation for the encouragement and support of Dr. Alexander J. A. Campbell, Director, the First (Tufts) Surgical Service, The Boston City Hospital; for the enthusiasm and efforts of Drs. Myron Segal and John Lynch, chief residents on our service; and for the helpful cooperation of Mr. Leon R. Nodine, vice-president of production, The Davol Rubber Company.

REFERENCES

1. Hampton, A. O.: A Safe Method for the Roentgen Demonstration of Bleeding Duodenal Ulcers, Am. J. Roentgenol. **38**: 565, 1937.
2. Schiff, L.: Hematemesis and Melena, Clinics **2**: 1,542, 1943-1944.
3. Schatzki, R.: Roentgen Demonstration of Esophageal Varices, Its Clinical Importance, Arch. Surg. **41**: 1,084, 1940.
4. Jones, C. M.: Diagnostic and Therapeutic Considerations of Gastro-intestinal Bleeding, New England J. Med. **235**: 773, 1946.

5. Allen, A. W.: Surgical Treatment of Duodenal Ulcer, Arch. Surg. **44**: 501, 1942.
6. Ritvo, M., Cotter, T. P., and Zamcheck, N.: Early Roentgen Diagnosis in Acute Bleeding From the Upper Gastrointestinal Tract. II. Roentgen Aspects, Am. J. Roentgenol. **66**: 728, 1951.
7. Zamcheck, N., Chalmers, T. C., Ritvo, M., and Osborne, M. P.: Early Diagnosis in Massive Gastrointestinal Hemorrhage, J. A. M. A. **148**: 504, 1952.
8. Chalmers, T. C., Zamcheck, N., Curtis, G. W., and White, F. W.: Fatal Gastro-intestinal Hemorrhage: Clinicopathologic Correlations in 101 Patients, Am. J. Clin. Path. **22**: 634, 1952.
9. Carter, M. G., and Zamcheck, N.: Esophagoscopy in Upper Gastrointestinal Bleeding, New England J. Med. **242**: 280, 1950.
10. Hinton, J. W., and Davis, D. A.: The Management of Massive Hemorrhage From the Gastrointestinal Tract Not Due to Peptic Ulcer, S. Clin. North America **29**: 317, 1949.
11. Hoerr, S. O., Dunphy, J. E., and Gray, S. J.: The Place of Surgery in the Emergency Treatment of Acute Massive Upper Gastrointestinal Hemorrhage, Surg., Gynec. & Obst. **87**: 338, 1948.
12. Meulengracht, E.: Fifteen Years' Experience With Free Feeding of Patients With Bleeding Peptic Ulcer. Fatal Cases, Arch. Int. Med. **80**: 697, 1947.
13. Kaplan, B.: Esophageal Varices, M. Rec. **154**: 176, 1941.
14. Barnet, C. A., and Cohen, S.: The Management of Massive Esophageal Hemorrhage With Tamponade and Thrombin, Gastroenterology **13**: 144, 1949.
15. Bennet, H. D., Baker, L., and Baker, L. A.: Complications in the Use of Esophageal Compression Balloons (Sengstaken Tube), Arch. Int. Med. **90**: 196, 1952.
16. Bixby, E. W., Jr.: Correspondence, J. A. M. A. **138**: 908, 1948.
17. Kenamore, R., and Elliot, G.: The Control of Esophageal Hemorrhage by Pneumatic Tamponade and Thrombin, Gastroenterology **13**: 73, 1949.
18. Linton, R. R.: The Emergency and Definitive Treatment of Bleeding Esophageal Varices, Gastroenterology **24**: 1, 1953.
19. Patton, T. B., and Johnston, C. G.: A Method for the Control of Bleeding From Esophageal Varices, Arch. Surg. **59**: 502, 1949.
20. Rowntree, L. G., Zimmerman, E. F., Todd, M. H., and Ajac, J.: Intra-esophageal Venous Tamponage. Its Use in a Case of Variceal Hemorrhage From the Esophagus, J. A. M. A. **135**: 630, 1947.
21. Sengstaken, R. W., and Blakemore, A. H.: Balloon Tamponage for the Control of Hemorrhage From Esophageal Varices, Ann. Surg. **131**: 781, 1950.
22. Reynolds, T. B., Freedman, T., and Winsor, W.: Results of the Treatment of Bleeding Esophageal Varices With Balloon Tamponage, Am. J. Med. Sc. **224**: 500, 1952.
23. Crohn, B. B., Marshak, R. H., and Galinsky, D.: Repeated Gastro-duodenal Hemorrhages Without Discoverable Explanation, Gastroenterology **10**: 120, 1948.
24. Jankelson, I. R.: Massive Hematemesis of Undetermined Origin, Gastroenterology **5**: 96, 1945.
25. Thompson, H. L., Oyster, J. M., Heid, J. B., and Morgan, F. M.: Hematemesis: A Study of Underlying Causes, Gastroenterology **7**: 320, 1946.
26. White, F. W., and Chalmers, T. C.: The Problem of Gross Hematemesis in a General Hospital. A Study of 400 Consecutive Cases, Trans. Assoc. Am. Phys. **61**: 253, 1948.
27. Stone, H. B.: Large Melena of Obscure Origin, Ann. Surg. **120**: 582, 1944.
28. Gray, H. K., Shands, W. C., and Thuringer, C.: The Problem of Massive Gastro-intestinal Hemorrhage From an Undetermined Source, Ann. Surg. **139**: 731, 1954.
29. Tocantins, L. M.: The Hemorrhagic Tendency in Congestive Splenomegaly (Banti's Syndrome). Its Mechanism and Management, J. A. M. A. **136**: 616, 1948.

DISSECTION OF THE HEPATIC PEDICLE AND RETROPANCREATICODUODENAL AREAS FOR CANCER OF THE STOMACH*

STUART W. ARHELGER, M.D., PAUL H. LOBER, M.D.,** AND
OWEN H. WANGENSTEEN, M.D., MINNEAPOLIS, MINN.

(From the Departments of Surgery and Pathology, University of Minnesota Medical School)

SINCE 1949, secondary or multiple operations called "second-look" procedures have been performed for selected cases of carcinoma of the stomach, colon, and rectum in which metastatic cancer in lymph nodes was found at the time of the primary operations. The interim results of these procedures have been reported elsewhere.[1, 3, 5, 7, 8] The "second-look" operations have offered a unique opportunity to study the nature and location of the spread of asymptomatic cancer following gastrectomy.

This report concerns itself with a study of the location of residual gastric cancer found at "second-look" operations and with a description and the preliminary results of the dissection of the hepatic pedicle and retropancreaticoduodenal lymphatic areas carried out at primary operations for cancer of the stomach.

Of 38 positive "look" operations for 26 patients with residual gastric cancer, the location of residual cancer could be anatomically identified and confirmed by histologic study at 30 operations. At 8 operations the metastases were diffuse (peritoneal seeding and multiple metastases to the liver) and no detailed tabulation of the site was made.

At 15 procedures for 11 patients of the 30 positive "look" operations for 26 patients, cancer was found within the lymph nodes, fibrous tissue, or fat of the hepatic pedicle (nodes and lymphatic channels of the ascending hepatic artery, portal vein, common bile duct, and cystic duct). Residual cancer was found in the retropancreaticoduodenal areas (superior or posterior retropancreaticoduodenal nodes) in 6 of the 30 operations. Fig. 1 illustrates the anatomic identification of residual cancer at 30 positive "look" operations.

The regular use of splenectomy to remove the regional lymphatics of the splenic pedicle, dissection of the juxtacardiac lymph nodes, and dissection of the splenic artery, celiac axis, and the aorta have been standard procedures in this clinic for several years.[3, 4, 6, 7] The "second-look" experience indicates need for an additional change in the plan of primary operations for gastric cancer to include a thorough dissection of the hepatic pedicle and the retropancreaticoduodenal lymphatics overlying the vena cava.

During the past eighteen months 24 primary extended lymphatic dissections including the hepatic pedicle and the retropancreaticoduodenal areas have been

Supported by the Damon Runyon Fund for Cancer Research and the Minnesota Division of the American Cancer Society.

Received for publication May 6, 1955.

*This report represents a portion of the senior author's thesis for the degree of Doctor of Philosophy in Surgery.

**Assistant Professor of Pathology.

performed. The operation has consisted of total or near total removal of the stomach, excision of the greater and lesser omenta, splenectomy, and dissection of the lymphatic channels and lymph nodes of the hepatic pedicle, the retropancreaticoduodenal area, the suprapancreatic areas along the horizontal hepatic artery and splenic artery, the celiac and aortic areas, and the abdominoparaesophageal and juxtacardiac chains. Fig. 2 illustrates the completed dissection.

A corollary pathologic study to determine the location and number of the lymph nodes removed, and the site and character of metastatic cancer contained in these specimens, has also been made.*

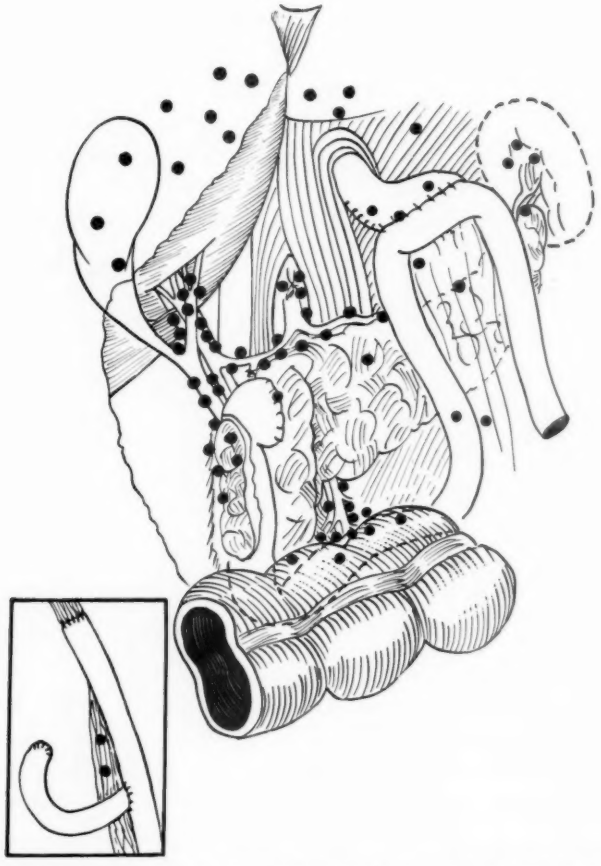


Fig. 1.—The anatomic identification of residual gastric cancer at 30 positive "second-look" operations. Each dot represents one instance of cancerous residual.

Twenty-two of the 24 patients had positive lymph nodes. Although many of these cases represent relatively advanced lesions, in each case the grossly detectable cancer was resectable.

It was interesting to find that 11 of the 24 specimens showed microscopic metastases to the lymph nodes of the hepatic pedicle. In the main these were found in the ascending hepatic artery chain or just medial to the portal vein.

*Thanks are acknowledged to Drs. James H. Casey, Dean M. Ring, and Donald B. Shahon, Fellows of the Department of Surgery, who, during an assignment in the Surgical Pathology Laboratory, gave this study helpful impetus by their careful dissections of the specimens included in this report.

However, several instances of involvement of the lymph nodes of the biliary structures (common duct, cystic duct) were encountered. Metastatic cancer in the retropancreaticoduodenal area (superior or posterior pancreaticoduodenal nodes) was found in 4 of the specimens.

In 9 of the 11 patients exhibiting metastases to the lymph nodes of the hepatic pedicle, the primary lesion was located in the pylorus or in the lesser curvature portion of the pylorus and in the body of the stomach (4 of these were limited to the pylorus, being either annular or on the lesser curve; the other 5 involved both the antrum and the body, and all of these were centered on the lesser curvature). One of the remaining 2 patients had a primary lesion involving the entire stomach, and in the other case there were two primary carcinomas: one involving the lesser curvature of the antrum and the body, and a second located at the cardiac orifice.

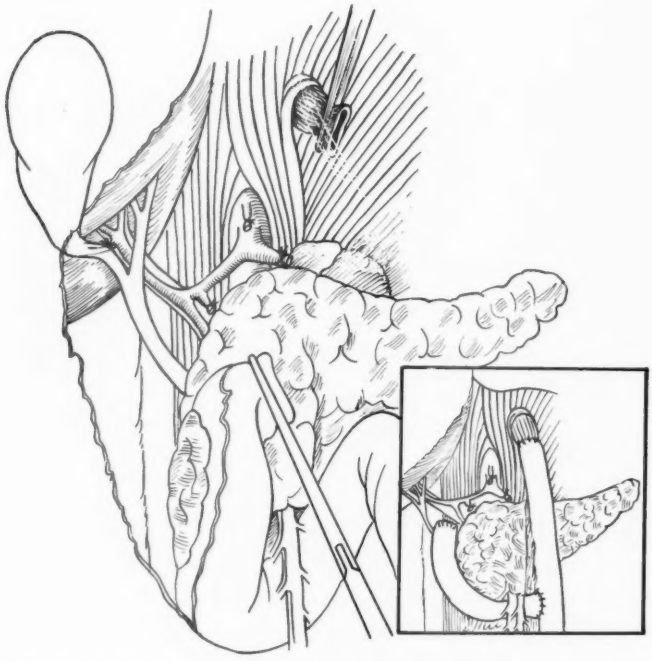


Fig. 2.—The extent of the completed lymphatic dissection for cancer of the stomach.

Of the 4 patients with metastatic cancer in the retropancreaticoduodenal lymph nodes, the primary lesions were limited to the pylorus in 2, and involved the pylorus and the body of the stomach in 2 cases. Two of these 4 patients exhibited extension of the carcinoma across the pyloric sphincter into the duodenum.

SUMMARY

The residuals found in "second-look" operations illustrate the presence of lymphatic metastases in areas anatomically designated as the lymphatics of the common, cystic, and hepatic ducts; the ascending hepatic artery lymphatic chain

of the duodenal hepatic ligament; and the retropancreaticoduodenal chains adjacent to the vena cava. These areas are not regularly dissected even at current radical primary operations.

Accordingly, a more complete dissection of the avenues of lymphatic drainage of the carcinomatous stomach has been devised with special emphasis upon the hepatic pedicle and retropancreaticoduodenal areas. Twenty-four operations of this type have been performed (in each case, all grossly detectable cancer was removed). A study of the surgical specimens revealed that 11 of the 24 patients had cancer within the lymphatic tissues of the hepatic pedicle. In 4 patients cancerous lymph nodes were present in the retropancreaticoduodenal area.

It is apparent that the lymph node dissection in present-day operations for cancer of the stomach is incomplete. An extension of the operation to include dissection of the hepatic pedicle and retropancreaticoduodenal area is recommended.

REFERENCES

1. Lewis, F. J., and Wangensteen, O. H.: Explorations Following Resection of the Colon, Rectum, or Stomach for Carcinoma With Lymph Node Metastases, Proc. Surg. Forum, Clin. Congress, Am. Coll. Surgeons, 1950, Philadelphia, 1951, W. B. Saunders Company, pp. 535-540.
2. Wangensteen, O. H.: The Problem of Gastric Cancer, J. A. M. A. **134**: 1161-1169, 1947.
3. Wangensteen, O. H.: The Surgeon's Approach to the Problem of Alimentary Tract Malignancy, Lancet **70**: 411-420, 1950.
4. Wangensteen, O. H.: Cancer of the Esophagus and the Stomach, Am. Cancer Soc. Monograph, 1951.
5. Wangensteen, O. H.: Further Experience Concerning the Principle of the "Second Look" Procedure in Alimentary Tract Cancer, Discussion before American Surgical Association, Ann. Surg. **132**: 561, 1950.
6. Wangensteen, O. H.: The Present Status and Likely Future Accomplishments of Surgery of Alimentary Tract Cancer, Proc. Second Nat. Cancer Conf. 84-103, 1954.
7. Wangensteen, O. H., Lewis, F. J., Arhelger, S. W., Muller, J. J., and MacLean, L. D.: An Interim Report Upon the "Second Look" Procedure for Cancer of the Stomach, Colon and Rectum and for "Limited Intraperitoneal Carcinosis," Surg., Gynec. & Obst. **99**: 257-267, 1954.
8. Wangensteen, O. H., Lewis, F. J., and Tongen, L. A.: The "Second Look" in Cancer Surgery, Lancet **71**: 303-307, 1951.

A METHOD FOR TOTAL REMOVAL OF BILE DUCT CALCULI

Preliminary Report

Julian A. Sterling, M.D., Sc.D., Philadelphia, Pa.

With the Technical Assistance of Bernard Diamond, A.B., Ong Tiong-Giok, M.D., and Harold Furman, M.D.

(From the Albert Einstein Medical Center)

A. INTRODUCTION

IN MANY cases, removal of all biliary tract calculi at operation is not always possible.[3] Even when their existence is recognized by cholangiography, their removal from the smaller hepatic ducts may not be feasible technically. In addition, there are undoubtedly calculi, particularly those in diverticula,[4] which are missed by all examinations. In such cases, a preferred definitive surgical procedure is to provide a wide adequate lumen through the duct termination or to establish a wide anastomosis between the duct and bowel.[5]

The use of a freshly prepared fibrin clot formed within the bile ducts has been studied. During its formation, the clot can enmesh intraductal biliary calculi. When the clot is firm, it may be removed together with the calculi. Dees[1, 2] had demonstrated the usefulness of a similar procedure to remove certain calculi from the renal pelvis. However, since fibrinogen is used to produce the clot, a risk of transmitting hepatitis is present unless (and perhaps even if) the fibrinogen is specially treated. The fibrinogen, now available, is prepared by ultraviolet irradiation against the transmissibility of hepatitis. This treatment of the fibrinogen, which may prohibit transmission of the virus, may also alter clotting capacity. Preliminary studies, therefore, were made to evaluate (a) the properties of treated fibrinogen in the formation of a fibrin clot; (b) the conditions attending the formation of the fibrinogen-thrombin coagulum; (c) the characteristics (and capabilities) of the firm coagulum, and (d) the practical application of this procedure with reference to calculi in the biliary tract.

It has been observed that the (new type of treated) fibrinogen, when mixed with thrombin, will form a clot after approximately one minute of contact. This clot becomes stronger and reaches optimal strength between three and thirty minutes after formation. The clot will dissolve under certain conditions. This procedure is applicable to calculi within the biliary tract. Its practical clinical applications have not yet been fully explored.

Received for publication April 25, 1955.

B. MATERIALS

All glassware and pipettes were chemically clean.

Fibrinogen.—(Human; ultraviolet irradiated; "Parenogen."*) One gram was slowly diluted with 50 c.c. of distilled water at 37.5° C. Complete clear solution was obtained within ten minutes. Final solution (neutral reaction) contained 0.02 Gm. per c.c. of fibrinogen.

Thrombin.—(Bovine; topical use.†) One thousand NIH units were dissolved first in 2 c.c., then diluted to 25 c.c. with 0.85 per cent sodium chloride. Final solution contained 40 NIH units per cubic centimeter.

Fig. 1.—Artificially produced fibrinogen-thrombin clot just removed from polyethylene tubing is seen to have enmeshed biliary calculi previously placed within the transparent tube.

Bile.—Human hepatic bile obtained from common duct drainage following surgery; no infection present. Bile used (1) full strength, and (2) diluted 1:10 with 0.85 per cent sodium chloride.

Varidase.‡—(Contains streptokinase [133,000 units] and streptodornase [150,000 units].) Diluted to 30 c.c. with 0.85 per cent sodium chloride. Final solution contained 4,000 units of streptokinase per cubic centimeter.

Diodrast.§—Sterile 35 per cent aqueous solution from 20 c.c. ampule.

Calcium Gluconate.—10 per cent solution in sterile 10 c.c. ampule.

Aerosol OT.—One drop of 10 per cent aqueous solution of dioctyl sodium sulfosuccinate was used as a wetting agent.

Calculi of varying sizes from the human biliary tract.

---

*Cutter Laboratories.
†Parke, Davis & Company.
‡Lederle Laboratories.
§Winthrop-Stearns, Inc.

C. PROCEDURES AND RESULTS

1. *Formation of the Clot.*—Various thrombin and fibrinogen solutions were mixed to determine the time of clot appearance and the proportions of each required to form the strongest clot.

Coagula began to appear within fifteen seconds of contact between these solutions (Fig. 1). Firm clot was present in most instances between sixty and eighty seconds. A clot forming after eighty seconds had high water content and was weak. The strongest clot was formed in a proportion of 2.5 volumes of fibrinogen to 2.0 volumes of thrombin (Table I). If less than 1.0 volume of

TABLE I. FIBRINOGEN CONSTANT VOLUME (2.5 ML.): THROMBIN VARIED

TOTAL VOL. (ML.)	THROMBIN (ML.)	0.85% NaCl OR H_2O OR BILE 1:10 (ML.)	FIRM CLOT FORMED (SECONDS)
5.0	2.5	- -	69
5.0	2.0	0.5	60
5.0	1.5	1.0	69
5.0	1.0	1.5	>80
5.0	0.5	2.0	>80

fibrinogen was present with 2.5 volumes of thrombin, a poor clot or no clot was formed (Table II).

TABLE II. FIBRINOGEN VARIED: THROMBIN CONSTANT VOLUME (2.5 ML.)

TOTAL VOL. (ML.)	FIBRINOGEN (ML.)	0.85% NaCl (ML.) OR H_2O (ML.) OR BILE 1:10 (ML.)	FIRM CLOT FORMED (SECONDS)
5.0	2.0	0.5	45
5.0	1.5	1.0	61
5.0	1.0	1.5	>80
5.0	0.5	2.0	>80
5.0	0.25	2.25	>80

2. *Lysis of the Clot.*—

a. *Varidase:* Continuing studies on clots which were formed in vitro indicated that 4,000 units of streptokinase in contact with a (5 c.c.—original volume) clot would dissolve the clot within six hours. In some instances, complete lysis of the clot did not occur until after twenty-four hours.

b. *Bile:* Hepatic bile prohibited formation of an adequate clot in 50 per cent of experiments. When hepatic bile was diluted ten volumes, with normal saline solution, clot formation was not altered. In some instances, there was delay in appearance of a clot when the fibrinogen-thrombin solution was in contact with either diluted or concentrated hepatic bile. In several instances, addition of concentrated hepatic bile caused lysis of the clot within eight to ten hours.

c. *Calcium gluconate:* Calcium gluconate did not alter the mechanism of fibrinogen-thrombin clot formation nor decrease the clot strength.

d. *Diodrast:* Diodrast caused the appearance of floccules within the fibrinogen-thrombin solutions and delayed clot formation. However, a solid clot formed after three minutes in the majority of studies.

e. *Other observations:* Fibrinogen solution which was five days old did not alter in its activity for clot formation (Table III). Fibrin clot did not form when the fibrinogen solution was more than ten days old.

In nearly all instances, spontaneous complete lysis of the clot was observed after twenty-four to thirty-six hours.

TABLE III. ACTION OF FIBRINOGEN FIVE DAYS OLD (CONSTANT VOLUME OF FIBRINOGEN EQUALS 2.5 ML.)

TOTAL VOLUME (ML.)	THROMBIN (ML.)	BILE 1:10 (ML.)	0.85% NACL (ML.)	CALCIUM GLUCONATE (ML.)	FIRM CLOT FORMED (SECONDS)
6.0	1.5	1.0	1.0	- -	54
6.0	2.0	1.0	0.5	- -	53
6.0	1.5	1.0	- -	1.0	52
6.0	2.0	1.0	- -	0.5	51

3. *Use of Clot in Biliary Tract (Necropsy).*—At necropsy, the biliary tract, from liver to duodenum, was warmed (water bath) to approximately 37.5 ° C. After washing the common bile duct with physiologic saline solution through the usual surgical approach, calculi were introduced. A "T" drain was sutured into the duct.[6] Eight to 10 c.c. of fibrinogen solution was introduced. Ten seconds later four to six c.c. of thrombin solution was added. Fifteen to twenty minutes after this, sutures were removed from the duct and the "T" drain was removed.

Fig. 2.—Several clots of different size contain biliary calculi.

In some instances, the coagulum was adherent to the duct and was freed by a thin metal probe passed around the duct wall. In a few instances, the coagulum did not adhere firmly to the "T" drain. It was then extracted by means of light traction with a forceps. Irrigation with salt solution by a small polyethylene catheter permitted removal of small portions of fibrin clot.

Even when the clot extended through the papilla and into the duodenum, it could be removed by gentle traction through the bile duct.

These studies were repeated using transparent tubes of $\frac{3}{32}$ and $\frac{9}{64}$ inches in diameter (Fig. 1). Visual evidence of the action by the fibrinogen-thrombin clot to enmesh the calculi was obtained. The clot, when removed from the transparent tubes, had enmeshed calculi at the extreme ends and had included calculi both at the periphery and within the thickness of the clot. (Figs. 2 and 3).

Fig. 3.—Close-up of fibrin clot reveals biliary sand, debris, and calculi within and attached to the clot.

D. DISCUSSION

To obtain rapid formation of the strongest clot at body temperature, 2.5 volumes of fibrinogen solution (0.02 Gm. per milliliter) were mixed with 2.0 volumes of thrombin solution (40 NIH units per milliliter). Under these conditions, the coagulum formed in approximately one minute with maximal tensile strength apparent after five to fifteen minutes. After five days in solution, fibrinogen will react to form a clot. However, its clot potentiality is lost after ten days.

Hepatic bile prevented clotting 50 per cent of the time. Diluted hepatic bile did not inhibit formation of fibrin clot. Calcium gluconate did not alter clot formation.

Varidase lysed clots in twelve to twenty-four hours. Fibrin clots formed in these experiments were dissolved spontaneously in thirty-six hours. The presence of Diodrast caused flocculation with delayed formation of a less firm clot.

Formation of a fibrin clot was possible in the human bile duct; calculi introduced into the duct were removable with the clot.

Use of this procedure has not yet been accomplished clinically.*

It was suggested† that it would be feasible, in certain cases, to prepare the patient's plasma fraction to contain fibrinogen which would be available at the time of choledocholithotomy and particularly for cases of recurrent calculi. This is presently under investigation.*

E. SUMMARY

1. Observations were recorded concerning the formation of a fibrin clot from solutions of fibrinogen (new type) and thrombin with respect to the optimal conditions of time, diluents, proportions of constituents, and additives.

2. Fibrin clot was produced in the human biliary ducts (necropsy) which enmeshed calculi. Removal of clot containing the calculi, en masse, was possible.

REFERENCES

1. Dees, John F., and Fox, H.: Properties of Human Fibrinogen Coagulum, J. Urol. 49: 503-511, 1943.
2. Dees, John F.: The Use of an Intrapelvic Coagulum in Pyelolithotomy, South. M. J. **36**: 167-174, 1943.
3. McAllester, A. J., and Call, D. W.: The Problem of Hepatolithiasis, Am. Surgeon 19: 695-707, 1953.
4. Sterling, J. A.: Diverticula of the Bile Duct, Am. J. Path. **25**: 325-338, 1949.
5. Sterling, J. A.: The Biliary Tract, Baltimore, 1955, Williams & Wilkins Company. In press.
6. Sterling, J. A.: The Use of "T" Drains in the Common Bile Duct, Surg., Gynec. & Obst. **100**: 762, 1955.

*Clinical use of fibrin clot for operative choledocholithotomy has been successfully and uneventfully accomplished in four cases (Aug. 12, 1955). The use of patients' plasma fraction as source for clot is presently impracticable because of insufficient tensile strength.

†By Drs. I. Young and H. Brody.

THERMAL BURNS IN MAN

IV. Body Weight Changes During Therapy

James D. Hardy, M.D.,* Eugene Jabbour, M.D.,** John R. Lovelace, M.D.,** William A. Neely, M.D.,** and Frank C. Wilson, Jr., M.D.,**
Memphis, Tenn.

(Department of Surgery and Surgical Laboratories, Medical College of the University of Tennessee, and the John Gaston Hospital)

Background and Purpose of Study

A MAJOR effect of the extensive thermal burn is the relatively enormous redistribution of water and electrolytes that is produced.[1] It has been estimated that as much as one-half of the extracellular sodium may be lost into the burned area, with an associated loss of extracellular water into the injured tissues as edema and into normal cells elsewhere in the body.[6, 7] In addition to these internal "losses" of extracellular fluid, transudation from the burned surfaces further depletes the body water and electrolytes.[9, 11] To maintain an adequate "effective" extracellular volume and thus to prevent shock due to diminished blood volume during the first forty-eight hours following the injury, an amount of fluid approximately equal to one-half the normal extracellular volume must be given to the extensively burned patient.[6, 7] This fluid appears to remain largely extracellular.[4, 8]

The purpose of the present series of studies was to determine the fate of the infused fluids. What amount of this fluid is temporarily retained? Are the losses through the wounds, unburned skin, lungs, and urine such as to maintain the patient in water balance, there being edema of the burned area but dehydration of the "normal" tissues elsewhere? Are the volumes of fluid currently administered to burn patients excessive, particularly in the older individuals among whom the mortality remains so great?

Our interest in the last question was sharply renewed by the clinical findings observed in a burn patient who was seen in consultation.† He was a husky, 31-year-old man who had been burned with hot tar when his truck overturned. In addition to a 40 per cent burn (30 per cent third degree), he had sustained a closed fracture of the left femur that had been successfully managed with an intramedullary nail and a fracture of the mandible that had been wired. We were asked to see him on the seventh day of hospitalization because of severe dyspnea, cyanosis, arterial hypertension, and generalized pulmonary râles which had developed the preceding day. A careful intake-output record had been kept, and it was noted that the combined intravenous and oral intake had exceeded

These studies were conducted under Army Contract No. DA-49-007-MD-296 between the University of Tennessee and the Office of the Surgeon General, Department of the Army.
Received for publication March 7, 1955.

*At present: Professor and Chairman, Department of Surgery, University of Mississippi, Jackson, Miss.
**Fellow in Surgical Research.

†This case has been cited elsewhere.[10]

the urinary output by 35 L. Since it was summer, with the room temperature rising to 102° to 104° F. each afternoon, the attending physician had urged salty fluids by mouth to combat the supposedly large daily fluid loss from the burned surfaces, the normal skin, and the lungs. However, the burn wounds, treated by the exposure method, had crusted over by the end of the second postburn day, and thereafter the insensible losses had presumably been confined largely to those from the unburned skin and lungs. Physical examination revealed extensive pitting edema that involved not only the areas adjacent to the burned surfaces but distant areas as well. It was clear that marked fluid retention had occurred. A drastic reduction of water and, more especially, salt intake, resulted in prompt improvement. The dyspnea, râles, hypertension, and cyanosis were relieved, and during the ensuing days the anasarca subsided as the urine volume exceeded the fluid intake each day, this in spite of the necessarily large daily insensible fluid loss through dissipation of body heat.

The findings in this patient prompted a review of the autopsy protocols of a number of burn deaths that had occurred at the John Gaston Hospital. The autopsies had been performed by members of the Department of Pathology of the Medical College of the University of Tennessee. It was found that pulmonary edema or other evidence suggesting excessive hydration had been a not uncommon finding in burn fatalities, and that the pathologist had often listed circulatory failure and/or pulmonary edema as a primary or secondary cause of death.

To answer the question of how much of the fluid infused in burn therapy is temporarily retained in the body, we undertook to weigh burn patients daily during the early phases of therapy. It was considered probable that the low urinary output commonly observed in these individuals during the first twenty-four to forty-eight hours following injury could be attributed in variable measure to the "alarming" effect of the injury upon the renal tubules, resulting in an increased rate of reabsorption of water. This effect is assumed to be mediated, at least in part, through an increased rate of secretion of the posterior pituitary antidiuretic hormone and of the hormones of the adrenal cortex. We felt that if these endocrine factors did constitute a significant cause of oliguria in the immediate postburn period, then the rate of urine formation might not be an entirely reliable guide to the state of body hydration.

It was assumed that large day-to-day weight changes would be due principally to variations in body water content. Hence, if the urine volume and the insensible fluid loss (including loss from the wounds) were not great, the patient would gain weight. If losses through the burn wounds were excessive, the individual would not gain weight and might actually lose weight, depending upon the fluid intake. As will be shown, patients with extensive burns, who necessarily received relatively large volumes of fluid, invariably gained weight during the first few days of therapy.

PROCEDURE AND RESULTS

Fourteen subjects whose burns exceeded 15 per cent of the body surface area were studied. Fluid therapy was usually administered according to the

formula* suggested by Evans.[5] By means of suitable rolling litter scales,[2] the turned patients were weighed immediately upon admission to the hospital and thereafter at frequent intervals. Wherever possible the exposure method was employed so as to avoid weighing errors due to saturated dressings. When the closed method was employed the patients were weighed when the dressings had been removed, although in such patients daily measurements were not always feasible.

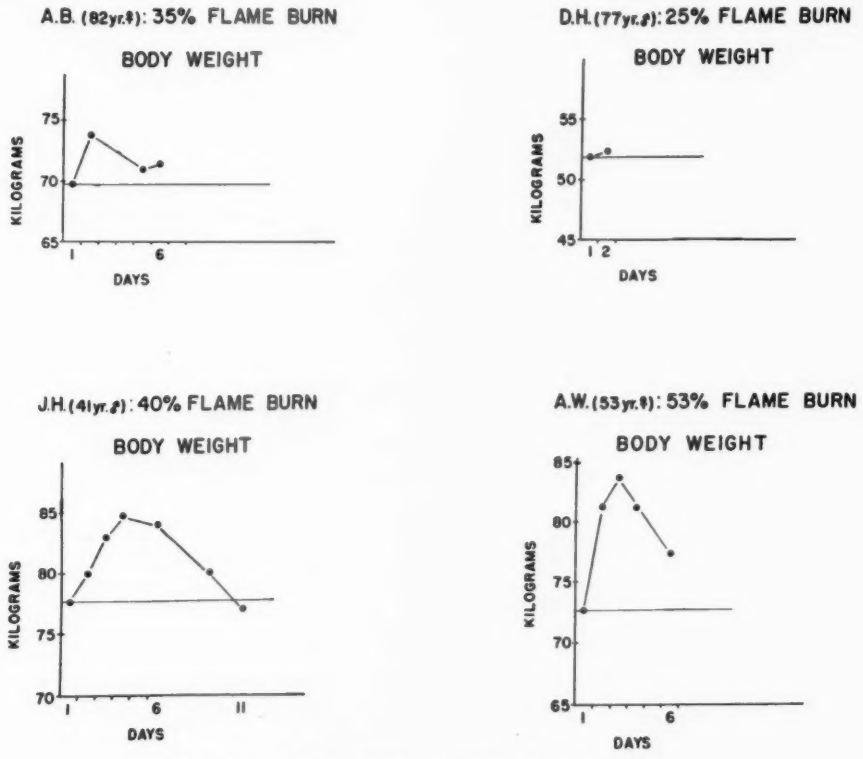


Fig. 1.—Body weight changes during therapy.

The results are presented in Figs. 1 to 4. It may be seen that all subjects gained weight during early therapy, and that those whose burns exceeded 40 per cent gained from 5 to 10 kilograms (22 pounds) during early fluid therapy. By Evans's formula, a patient weighing 70 kilograms whose burns covered 40 per cent of the body surface area received between 7 and 8 L. of fluid during the first twenty-four-hour period following the injury and approximately one-half this amount during the second twenty-four hour period. Thereafter, the fluid administered was based upon clinical and laboratory findings.

The greatest weight increases were exhibited by patients R. J., A. Ni., and A. W. Each of these individuals gained approximately 10 kilograms. All

*First 24 hours: Blood, plasma, or plasma expander = 1 c.c. × % burn × body weight in kg.; isotonic saline = 1 c.c. × % burn × body weight in kg.; glucose solution = 2,000 c.c.
Second 24 hours: One-half the volume of colloid and of saline calculated for first 24 hours, plus 2,000 c.c. of glucose.
Thereafter: As judged necessary on general clinical grounds.

had extensive burns that were eventually fatal but, by and large, the excess fluid appeared to have been lost from the body by the time of death and the death was not considered to be due to overhydration. In point of fact, in

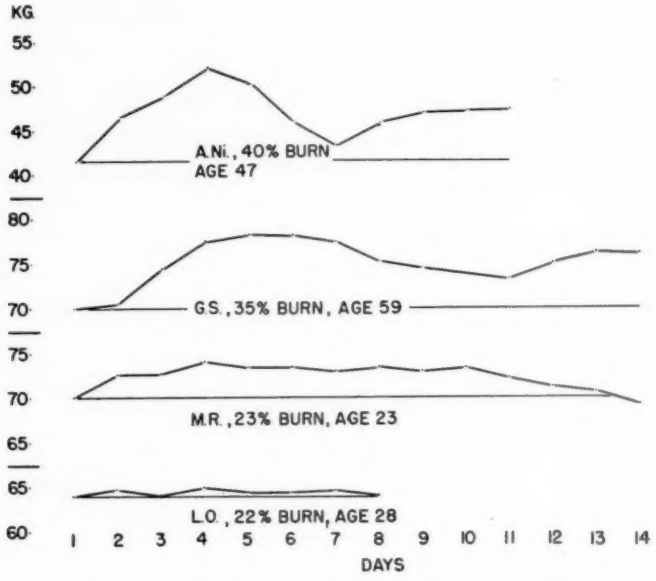


Fig. 2.—Body weight changes during therapy.

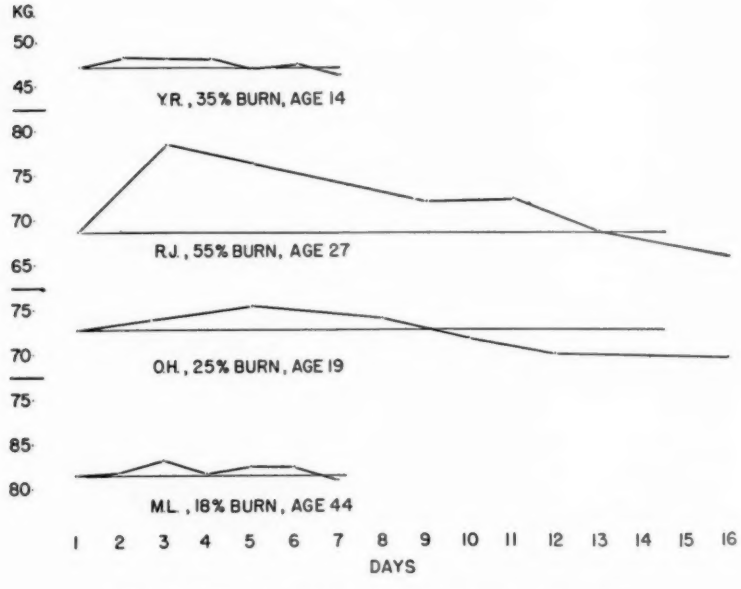


Fig. 3.—Body weight changes during therapy.

two extensively burned patients whom we have studied, the fluid therapy (blood, plasma, saline, glucose, and, where necessary, sodium lactate) was given only as needed to maintain a normal blood pressure; yet, the volume

actually required rather closely approximated that which had been estimated by Evans's formula. The weight gain ranged from 5 to 12 kilograms in those subjects whose burns exceeded 40 per cent of the body surface area.

In the more extensively burned individuals the body weight began to diminish on from the fourth to the seventh postburn day, and this process was usually complete by the end of the thirteenth day. This decline in body weight was concomitant with (1) a decrease in fluid intake, (2) an increase in urine volume, (3) a negative nitrogen balance, and, rarely, (4) an apparent increase in "insensible fluid loss."

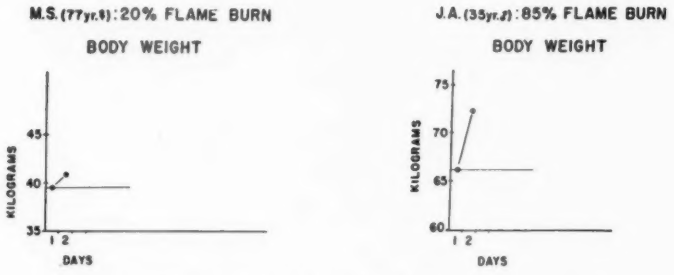


Fig. 4.—Body weight changes during therapy.

Relation of Weight Gain to Percentage of Surface Area Burned.—Inspection of Figs. 1 to 4 reveals that there was a positive correlation between the degree of weight gain and the extent of the burn. This resulted from the fact that the greater the percentage of the body surface area burned, the greater the volume of fluid administered during early therapy. The fluid losses from the burned surfaces can be relatively small, even in the extensively burned individual. Therefore, since the urinary output in the immediate postburn period may be no greater in the patient who receives a large volume of fluid than in the patient who receives only a modest volume (indeed, the urinary output in the former may at times be less, for several reasons), it is evident that, barring excessive insensible fluid losses, the extensively burned individual will inevitably gain considerable weight during therapy. The need for additional fluid in the extensively burned individual is, of course, due to the fact that a greater amount of fluid is "sequestered" in the tissues beneath the burned skin and elsewhere. The increased need for blood as such is due to dilatation of capillaries, stasis, hemolysis, and increased loss through injured capillary walls. It was shown experimentally by Netsky and Leiter[12] that there is an increase in capillary permeability to protein in the burned animal, and these findings were extended by Cope and Moore[3] using radioactive colloids. There was virtually no evidence that capillary permeability was increased in areas remote from the burn.

Insensible Fluid Loss.—The diphasic weight curve exhibited by patient A. Ni. (Fig. 2) is of particular interest. The decline in the level on the seventh day coincided with a sharp increase in "insensible fluid loss."* On these

*The approximation for insensible fluid loss was obtained by substituting in the following formula: Fluid intake equals body weight change plus urine output plus insensible fluid loss.

particular days, when the estimated insensible fluid loss was almost 6 L., the urine volume ranged from 500 to 1,000 c.c. daily. Studies to be reported have indicated that insensible fluid losses may vary greatly from one patient to another and in the same patient from day to day.

Loss of Body Tissue.—It is to be noted that the weight curve for patient R. J. (Fig. 3) had returned to the admission base line by the fourteenth postburn day but then continued steadily downward. Although not shown on the chart, by the thirtieth postburn day he had lost 25 pounds from the admission weight. Since he was not an obese individual, most of this marked weight loss was due to lean tissue loss, and his physical appearance substantiated this conclusion. Thus, since the third postburn day, when his weight was 25 pounds above the admission value, there had been a weight fluctuation of 48 pounds. Every effort was made to tube-feed this individual successfully, but all measures were ineffective.

DISCUSSION

It is clear that the extensively burned patient who is given adequate fluid therapy will almost invariably gain weight. This results both from the large amounts of fluid which are necessary to combat shock and the fact that the intense trauma of the burn injury evokes hormonal changes that may diminish the excretion of water by increasing renal tubular reabsorption. Consequently, the rate of urine flow, although a valuable guide, unquestionably suffers from definite limitations as an indication of the state of body hydration during the first forty-eight hours following the burn.

SUMMARY AND CONCLUSIONS

1. Body weight changes in fourteen patients whose burn exceeded 15 per cent of body surface area are presented. Evans's formula was employed as a guide to fluid therapy.

2. All extensively burned subjects exhibited a considerable gain in body weight during early fluid therapy. The weight increase in patients whose burns exceeded 40 per cent ranged from 5 to 12 kilograms. After the first two weeks the body weight had almost always declined to the admission level, and thereafter a major decline was commonly observed in the more severe injuries.

3. The volume of "insensible fluid loss" from the lungs, normal skin, and the burned surfaces appears to be highly variable but is influenced by the extent of the burn.

4. The volume of fluid actually required to sustain the blood pressure in two extensively burned individuals was found to be in close agreement with the volume estimated by Evans's formula. This was particularly true in respect to colloid materials required.

5. While valuable, the rate of urine flow suffers from definite limitations as a guide to fluid therapy in the early postburn period.

REFERENCES

1. Blalock, A.: Experimental Shock. VII. The Importance of the Local Loss of Fluid in the Production of the Low Blood Pressure After Burns, Arch. Surg. **22**: 610, 1931.
2. Bluemle, L. W., and Elkinton, J. R.: A Stretcher-scale for the Accurate Weighing of Bedridden Patients, J. Lab. & Clin. Med. **41**: 300, 1953.
3. Cope, O., and Moore, F. D.: A Study of Capillary Permeability in Experimental Burns and Burn Shock Using Radioactive Dyes in Blood and Lymph, J. Clin. Invest. **23**: 241, 1944.
4. Cope, O., and Moore, F. D.: The Redistribution of Body Water and the Fluid Therapy of the Burned Patient, Ann. Surg. **126**: 1010, 1947.
5. Evans, E. I., Purnell, O. J., Robinett, D. W., Batchelor, A., and Martin, M.: Fluid and Electrolyte Requirements in Severe Burns, Ann. Surg. **135**: 804, 1952.
6. Fox, C. L., Jr., and Baer, H.: Redistribution of Potassium, Sodium and Water in Burns and Trauma, and Its Relation to the Phenomena of Shock, Am. J. Physiol. **151**: 155, 1947.
7. Fox, C. L., Jr., and Keston, A. S.: The Mechanism of Shock From Burns and Trauma Traced With Radiosodium, Surg., Gynec. & Obst. **80**: 561, 1945.
8. Hardy, J. D., Lovelace, J. R., Jabbour, E., and Bramlitt, E. E.: Thermal Burns in Man. VI. Body Fluid Compartments During Early Therapy. To be published.
9. Hardy, J. D., Neely, W. A., and Wilson, F. C., Jr.: Thermal Burns in Man. VII. Insensible Fluid Loss, SURGERY **38**: 692, 1955.
10. Hardy, J. D., Neely, W. A., Wilson, F. C., Jr., Milnor, E. P., and Wilson, H.: Fluid Kinetics Following Thermal Burns in Man, SURGERY **34**: 457, 1953.
11. Moore, F. D., Langohr, J. L., Igebretsen, M., and Cope, O.: The Role of Exudate Losses in the Protein and Electrolyte Imbalance of Burned Patients, Ann. Surg. **132**: 1, 1950.
12. Netsky, M. G., and Leiter, S. S.: Capillary Permeability to Horse Proteins in Burn-shock, Am. J. Physiol. **140**: 1, 1943.
13. Rosenthal, S. M.: Experimental Chemotherapy of Burns and Shock. III. Effects of Systemic Therapy on Early Mortality, Pub. Health Rep. **58**: 513, 1943.

THERMAL BURNS IN MAN

VII. "Insensible Fluid Loss"

James D. Hardy, M.D.,* William A. Neely, M.D.,** and
Frank C. Wilson, Jr., M.D.,** Memphis, Tenn.

(From the Department of Surgery and Surgical Laboratories, Medical College of the University of Tennessee, and the John Gaston Hospital)

A MAJOR source of uncertainty in estimating the fluid requirements following thermal burns in man has been the relative paucity of information regarding the actual fluid losses through the unburned skin, lungs, and the burn wounds. Cope and Moore,[1] through laborious analyses of the electrolyte and nitrogen content of the dressings and bedclothes, reported approximations for the water and electrolyte losses from the burned surfaces. As might have been expected, the wound fluid losses exhibited a direct correlation with the extent of the burn. It was found that during the first forty-eight hours an average of 50 c.c. (seven patients) was lost for each per cent of surface area burned. These workers also employed such measurements as the fluid intake, the change in "extracellular" space (measured with thiocyanate), and the urine output to estimate indirectly the external fluid losses. A major source of potential error, however, was the possible entry of water into the cells; such a circumstance would have resulted in an erroneously large estimated external water loss. Although their patients were not weighed, the data obtained by Cope and Moore are in general agreement with those that we have obtained using frequent body weight measurements.

As a part of a general investigation of fluid kinetics following thermal burns in man, the insensible fluid losses were estimated in seven patients whose burns involved more than 15 per cent of the body surface area. Daily body weight determinations were made. The study did not distinguish between the losses from the burn surfaces and the insensible fluid losses from the unburned skin and the lungs. Measurements were complete in five patients, and these data form the basis of the present report. It has been found that the "insensible fluid loss" may vary greatly from one patient to another and even in the same patient from day to day.

PROCEDURE AND RESULTS

The patients were weighed immediately upon admission to the hospital before fluid therapy had been started and then daily thereafter in most instances.[2] Where the open method of treatment could not be used the patients

These studies were conducted under Army Contract No. DA-49-007-MD-296 between the University of Tennessee and the Office of the Surgeon General, Department of the Army.
Received for publication April 4, 1955.

*Present address: Hospital of the University of Mississippi, Jackson, Miss.
**Fellow in Surgical Research.

were weighed with the dressings removed, but daily weight measurements in the case of those treated by the closed method were not usually feasible. A careful record of the fluid intake and the urine output was kept. Since the patients consumed little food during the period of study, the water derived from food oxidation did not introduce a significant source of error. None had diarrhea. The values for "insensible fluid loss" were estimated by substituting in the following formula: Fluid intake = body weight change + urine output + insensible fluid loss. Three of the four values were known, thus allowing computation of the unknown, insensible loss. This formula, as noted, ignored body weight changes due to tissue metabolism, but the loss of body tissue each twenty-four hours was of small magnitude when compared with the relatively large changes in water exchange which occur during early burn therapy.

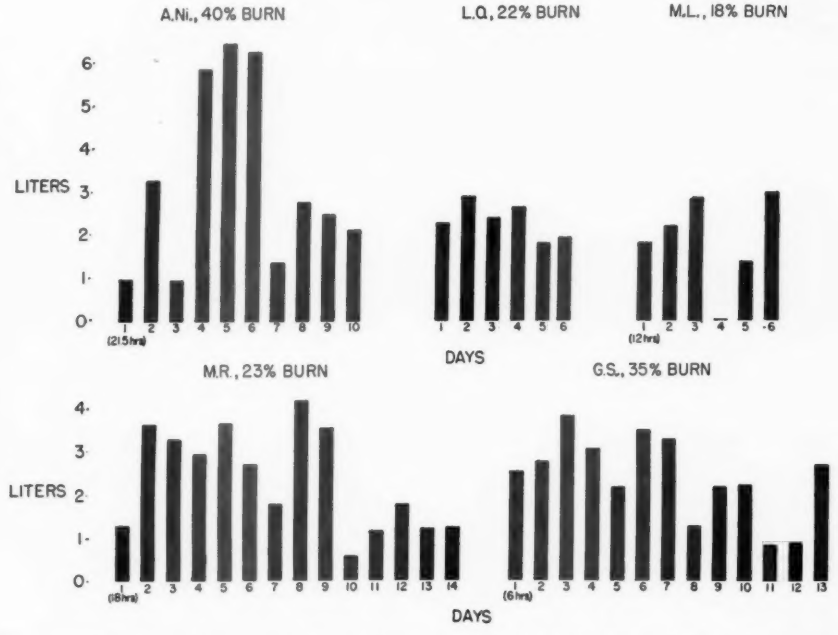


Fig. 1.—It will be apparent that the "insensible fluid loss" from the lungs alone in Patient M. L. would have exceeded the volume recorded on the fourth day. Thus, the method by which these data were obtained has definite limitations. Nevertheless, in general, the values presented are sufficiently dependable to emphasize the possible magnitude and the variability of "insensible loss" and its important influence upon fluid requirements in burns.

The results are shown in Fig. 1. All patients were studied during the fall or winter months, excessive ambient temperatures being thus avoided. Patient A. Ni. exhibited on days 4, 5, and 6 the greatest insensible fluid loss observed. During these days the body weight (upon which measurement the value for insensible loss was, of course, based) declined sharply from the maximum rise that had occurred during the first three days of therapy; thereafter, the weight curve rose again. The urine volume ranged from 500 to 1,000 c.c. daily throughout this time. Two other patients, whose measurements were incomplete and are thus not reported here, also exhibited as much

as 5 L. of insensible fluid loss on isolated days. Otherwise, the "insensible loss" (from lungs, normal skin, and burn wounds) tended to be in the general range of from 2 to 3 L. in the patients shown, diminishing to the normal level of from 1 to 1.5 L. as the burn wounds crusted over.

DISCUSSION

Several tendencies have been noted. First, patients treated by the exposure method appear to lose less fluid by "insensible loss" after the wounds have crusted over. Furthermore, preliminary data suggest, but do not clearly establish, that the insensible losses are less when the wounds are treated by the open method and allowed to crust over than when the wounds are covered with bulky dressings which become saturated with transudate but which prevent crusting. Second, the burn may "weep" very little during the first or even the second day, only to exude copious amounts of serum thereafter. The extreme in this respect was a patient who was burned with boiling lye. On admission, the desquamated areas were weeping little if at all, but after approximately five days all exposed areas began to weep copiously. Third, an improved state of hydration has appeared to be associated with an increased loss from the burned surfaces. This statement is based upon the fact that the wounds of two patients, whose results are not shown here, began to weep freely after fluids had restored the normal blood pressure and urine flow. Fourth, as noted by Cope and Moore, the insensible loss was in a measure directly proportional to the extent of the burn.

SUMMARY AND CONCLUSIONS

1. Approximations for insensible fluid losses in five burn patients are reported. The "formula" employed was: Fluid intake = body weight change + urine output + insensible loss.

2. The apparent insensible fluid loss varied markedly in different patients and often in the same patient from day to day. The insensible loss was, as might be expected, greatest during the early days of the burn. The largest daily insensible fluid losses observed were on the order of magnitude of 6 L., although this level was never long sustained.

3. In patients treated by the open or exposure methods, the insensible fluid losses appeared to diminish after the wounds had crusted over. Moreover, weeping from the burned surfaces perhaps diminished more promptly if a good crust was obtained with the exposure method than if the wounds were covered with dressings.

4. There was evidence to suggest a positive correlation between the degree of body hydration and the fluid loss from the burned surfaces during the first twenty-four hours.

5. There was a positive correlation between the per cent of body surface burned and the "insensible fluid loss."

6. It is concluded that the "insensible fluid loss" constitutes a variable of much clinical significance in the fluid therapy of burned patients.

REFERENCES

1. Cope, O., and Moore, F. D.: The Redistribution of Body Water and the Fluid Therapy of the Burned Patient, Ann. Surg. **126**: 1010, 1947.
2. Hardy, J. D., Jabbour, E., Lovelace, J. R., Neely, W. A., and Wilson, F. C., Jr.: Thermal Burns in Man. IV. Body Weight Changes During Early Therapy, SURGERY **38**: 685, 1955.

THE MECHANISM OF DEATH AFTER RESUSCITATION FOLLOWING ACUTE CIRCULATORY FAILURE

JACK W. CROWELL, M.S., GUY P. SHARPE, M.D.,* ROBERT L. LAMBRIGHT, M.D.,* AND WILLIAM L. READ, M.D.,* JACKSON, MISS.

(From the Department of Physiology and Biophysics, University Medical Center, Jackson, Miss.)

IT HAS been generally accepted that the central nervous system cannot withstand periods of circulatory arrest greater than three to five minutes at normal body temperature without the occurrence of permanent cerebral damage.[1] Experimental animals subjected to periods of circulatory arrest and patients in whom cardiac arrest occurs die more frequently than they live even when the periods of arrest are no longer than 2 to 3 minutes.[2, 3] It is also generally believed that this cerebral damage is due primarily to the lack of oxygen.[4] By recording changes in blood coagulation time during and following circulatory arrest, it has been possibly, as reported in the present paper, to show that another mechanism may be responsible for the cerebral damage and high mortality rate following periods of acute circulatory failure.

METHODS

Adult mongrel dogs were confined one day prior to operation and were given adequate water but no food. After anesthetization with 30 mg. per kilogram of sodium pentobarbital, an endotracheal tube was inserted and the chest was opened by the technique of MacQuigg[5] at the sixth rib. After allowing a few minutes for bleeding to stop, the heart was fibrillated by applying a stimulus of 60 cycles per second. As soon as fibrillation was definitely established, the artificial respiration was also stopped, and this was considered to be the beginning of circulatory arrest. Following a given period of time, artificial respiration was resumed, and the heart was massaged and defibrillated a few minutes later by 110 volts alternating current countershock. The beginning of massage was considered to be the end of circulatory arrest. The results were compared between control dogs and dogs heparinized before fibrillation. As soon as circulation was re-established, the wound was repaired.

Blood coagulation times were determined at frequent intervals for long periods of time. To do this, it was necessary to develop a method utilizing an easily obtainable, small sample with a rapid end point. This was accomplished by placing a polyethylene catheter into the inferior vena cava via the femoral or saphenous vein. An individual sample was taken by (1) withdrawing blood from the catheter until venous blood was obtained, (2) attaching a second syringe

This investigation was supported by a research grant-in-aid from the National Heart Institute, National Institutes of Health, United States Public Health Service.

Received for publication April 11, 1955.

*Fellow in Surgery, The Vicksburg Hospital and Clinic, Vicksburg, Miss.

to the catheter, and (3) removing 1.5 ml. of blood. One milliliter of the blood was placed in an 8 mm. dry glass tube and then placed in a 37° C. water bath. The samples were inverted every 30 seconds, and the coagulation time was recorded as time elapsed between the blood entering the syringe and the time at which the tube could be inverted without spilling its contents. The catheter was flushed with saline solution before and after each determination.

A permanently inserted polyethylene tube for obtaining the blood samples was used for the following reasons: (1) studies were desired on mixed venous blood, not blood returning from a given area; (2) as many as 60 samples of blood were taken at rapid intervals; and (3) small blood clots which were present in the mixed venous blood could not be obtained by needle puncture; thus, a large bore catheter was used. Control values for this method remained extremely constant for many successive determinations, and these are shown in each individual experiment, particularly for the animals subjected to a very short period of circulatory arrest. The important consideration in the present study is mainly the change in coagulation time rather than the absolute coagulation time.

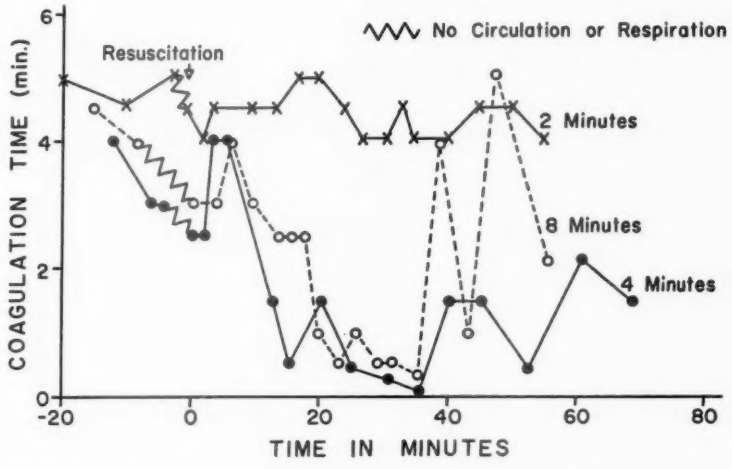


Fig. 1.—The changes in coagulation time following resuscitation.

RESULTS

Effect of Circulatory Arrest Upon Coagulation Time.—Figs. 1 and 2 show the results from a series of 4 dogs in which circulatory arrest lasted 2, 4, 6, and 8 minutes. Zero time on the abscissa indicates the beginning of resuscitation, and the wavy line indicates the period of circulatory arrest. Resuscitation usually was completed within 4 minutes. The control values are shown for all 4 dogs and range from 3 to 5 minutes, the approximate range expected during surgery. Note in particular the coagulation time of the dog subjected to only 2 minutes of circulatory arrest. Although some minor variations in all the coagulation times exist, these are within the limits expected when tubes are inverted every 30 seconds, and in no case did the coagulation vary more than 30 seconds from the control value. This animal lived.

When the period of circulatory arrest was lengthened, the coagulation time following resuscitation was entirely different from that in the previously described animal. Immediately after resuscitation the coagulation time usually increased by a slight and probably insignificant amount. After this, however, it became erratic, and the blood was very hypercoagulable. Within 20 to 40 minutes, the coagulation time was often 30 seconds or less. However, it rose again after this period of hypercoagulability. Note in particular the dog subjected to 8 minutes of circulatory arrest. After the extreme hypercoagulable phase, there were two samples within the normal range interspersed among 3 samples that clotted very rapidly; this illustrates how erratic the coagulation times became.

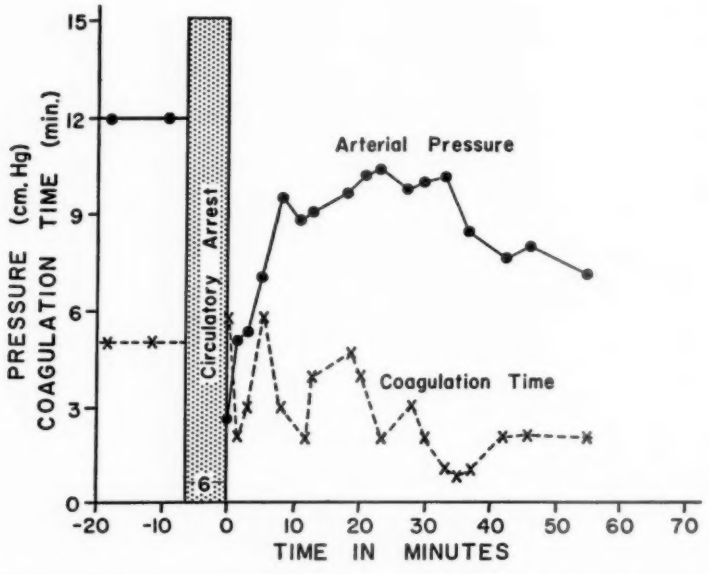


Fig. 2.—The correlation of coagulation time and arterial pressure.

Correlation of Coagulation Time With Femoral Blood Pressure.—To appreciate the significance of this erratic coagulation, in Fig. 2 the coagulation time was plotted on the same graph as the femoral arterial pressure. Again the pattern of erratic coagulation times developed; however, it is evident in this graph that after the first period of hypercoagulability there was a temporary decrease in arterial pressure for every instance in which the coagulation time decreased; finally, when the coagulation time reached its lowest point, the arterial pressure began an "irreversible" decline, and the animal was in a type of irreversible shock. Animals showing this type of coagulation curve died almost invariably.

Effect of Asphyxia on Coagulation Time.—A possible reason why the coagulation time changes and why these changes occur approximately 30 minutes after resuscitation is indicated by Fig. 3. This experiment was similar to those given previously but differed in that respiration was stopped for 10 minutes, and the heart was not fibrillated. This dog was given 2 mg. of heparin per kilogram of body weight 5 minutes before cessation of artificial respiration. Follow-

ing the onset of asphyxia, the blood pressure rose and then fell as the heart became too anoxic to propel blood. At this point, cardiac massage was instituted until the cardiac muscle was reoxygenated and the heart resumed its function.

The coagulation time, shown by the dashed line, fell very rapidly during the period of asphyxia, indicating that either some substance was entering the blood stream or some condition existed which was antagonistic to the heparin. Following the re-establishment of respiration, the coagulation time rose again. However, there appeared to be present in the blood a large quantity of fibrin and small emboli, and these settled and adhere together to form a visible clot, as noted in Fig. 3. This clot became present before the entire volume of blood coagulated, and there was a difference of several hours in some of the tubes. Thus, it seemed that during the period of asphyxia a large quantity of minute

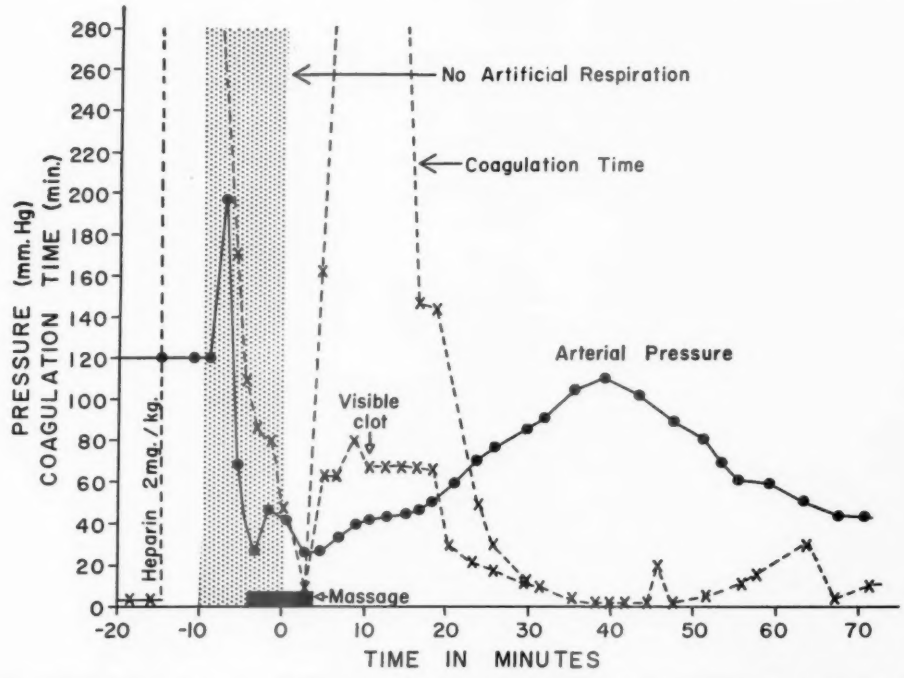


Fig. 3.—The effect of ten minutes of asphyxia on the coagulation time of a heparinized dog.

blood clots formed somewhere in the circulatory system, and these were later released. Note in particular that as the coagulation time decreased to near zero, at this exact point on the graph the arterial pressure, previously rising, began to fall.

Autopsies Following Death of the Resuscitated Animals.—The hearts and lungs of 27 animals given no heparin and of 20 animals given 2 mg. per kilogram of heparin were examined grossly for the presence of emboli. All of the animals had been subjected to 10 minutes of circulatory arrest.

In every case it was possible to detect emboli in the lungs, although it appeared that most of the emboli were extremely small and barely visible to the unaided eye. Many larger clots were found, however, particularly in those dogs that lived for several hours.

Survival After Circulatory Arrest of Nonheparinized and Heparinized Dogs.—Thirteen dogs were subjected to 5 minutes of circulatory arrest without the prophylactic use of heparin. Of the 13 dogs, 2 could not be resuscitated, 8 were resuscitated but later died, and 3 lived permanently, although one of the permanent survivors suffered loss of vision. Most of the resuscitated dogs had a period of hyperexcitability about 6 to 15 hours after resuscitation and also became extremely hyperthermic. Following this was a period of progressive depression from which they did not recover.

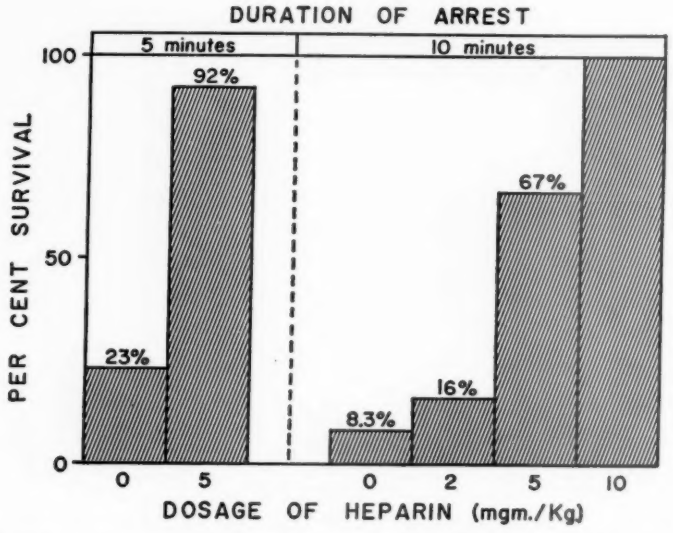


Fig. 4.—The effect of prophylactic heparinization on survival after fixed periods of circulatory arrest.

An additional 13 dogs were subjected to 5 minutes of circulatory arrest after the injection of 5 mg. per kilogram of heparin. Twelve of these animals lived, and only one showed a slight intention tremor for five days. One animal died apparently as a result of vomitus. This group of animals did not show the hyperexcitability and hyperthermia characteristic of the previous group. The heparin was counteracted with 7.5 mg. per kilogram protamine sulfate after resuscitation.

Twelve dogs were subjected to 10 minutes of circulatory arrest without the prophylactic use of heparin. Only one of these dogs survived, and this animal suffered severe cerebral damage. The postoperative course of the remaining animals was similar to that of the 5-minute, nonheparinized group.

Additional animals were subjected to 10 minutes of circulatory arrest with varying doses of heparin. Of 24 animals given 2 mg. per kilogram, 4 survived, and 2 of these suffered permanent cerebral damage. Three animals were given 5 mg. per kilogram of heparin, and 2 of these survived permanently with no cerebral damage. Four animals were given 10 mg. per kilogram of heparin, and all 4 survived permanently; however, 1 of these had transient loss of vision. Protamine sulfate was used to counteract the heparin in all cases. The maximum

period of arrest from which permanently normal survival was obtained was 10.8 minutes. However, it appears that, if the bleeding difficulties associated with larger dosages of heparin are solved, even longer periods may be tolerated.

Fig. 4 is a summary of all experiments.

DISCUSSION

Many attempts have been made in the past to determine the amount of time the central nervous system can withstand circulatory arrest without permanent damage. However, because many methods do not cause total cerebral ischemia, only those in which circulatory arrest was produced by stopping the heart will be considered in the present discussion.

Batelli[6] appears to be the first person to use the method of fibrillation and defibrillation, and he was able to resuscitate the hearts of cats after 15 minutes of fibrillation. However, in a summary of all experiments, there was not one single permanent survivor when the period of circulatory arrest was 5 minutes or longer. Using a similar method on dogs, Hooker[7] was unable to obtain any survivors after 4 minutes or more of fibrillation, and a summary of his experiments shows that between ½ minute and 5 minutes, only 60 per cent were resuscitated, and only 27 per cent are listed as permanent survivors. Stearns,[8] also using this method, had only a 36 per cent permanent survival after 5 minutes of fibrillation even though the heart was massaged during the five-minute interval. Thus, the control values for the series of dogs subjected to 5 minutes of complete arrest in the present study are comparable to, if not somewhat better than, those reported by others, for the permanent survival rate was 23 per cent.

In direct contrast to this, when the animals were heparinized before the 5-minute period of fibrillation, the survival rate increased to 92 per cent, with only one fatality due directly to vomitus. Resuscitation was much easier in these animals than in control animals, and not one single failure to resuscitate was recorded in any of the heparinized dogs as compared to the high rate of failures to resuscitate in the unheparinized dogs.

Only the paper by Batelli[6] listed the results in animals subjected to 10 minutes of complete arrest, and, as already mentioned, there were no survivors. In our control series, 25 per cent of the animals were not resuscitated, and only 1 of the 12 animals survived. Again, when the animals subjected to 10 minutes' arrest were heparinized before fibrillation, there were no failures at resuscitation, and many permanent survivors were obtained; with the larger dosages of heparin, these animals survived with no permanent cerebral damage, although some of them suffered transient damage.

If the degree of cerebral destruction were determined by the phylogenetic structure of the brain, then it would be logical to assume that a given period of circulatory arrest would allow degeneration of certain portions of the brain, but in all cases the animal would live until such time as the arrest were of sufficient duration to affect the vital centers. This phylogenetic theory of cerebral destruction has not been conclusively confirmed, and, indeed, some pathologic evidence is to the contrary.[9-11]

It appears, therefore, that it is not necessarily the central nervous system that imposes the limiting factor in the duration of arrest tolerated by these experimental animals, but, instead, it is the development of small blood clots in the vascular system. As demonstrated in the figures at the beginning, these small blood clots form in the vascular system during periods of circulatory arrest or during periods of extreme anoxia. However, it is approximately 30 minutes later that the majority of them are washed out to block the flow of blood through the pulmonary system. At this time the arterial pressure begins to fall; thus, there results a type of "irreversible" shock.

It also appears that some of the central nervous system destruction formerly attributed to the primary effects of anoxia may be due to the formation of these small clots in the central nervous system. Indeed, cerebral damage caused by the injection of coagulants has been reported by Hoefer.[12]

SUMMARY

1. By fibrillating and defibrillating the heart, studies have been made on the cause of death following brief periods of circulatory arrest.

2. Minute blood clots have been detected in the circulatory blood and also have been noted in the lungs. If the period of circulatory arrest is greater than 3 to 5 minutes, a large number of these small clots form and cause death of the animal.

3. If the animals are heparinized during the period of circulatory arrest, the mortality rate is greatly decreased, and the duration of arrest may be extended to approximately twice that of a nonheparinized animal.

4. The data presented show that the central nervous system is not as sensitive to anoxia as previously believed.

REFERENCES

1. Weinberger, L. M., Gibbon, M. H., and Gibbon, J. H., Jr.: Temporary Arrest of the Circulation to the Central Nervous System, Arch. Neurol. & Psychiat. **43**: 615, 1940.
2. Cole, F.: Cardiac Massage in the Treatment of Arrest of the Heart, Arch. Surg. **64**: 175, 1952.
3. Dale, W. A.: Cardiac Arrest, Ann. Surg. **135**: 376, 1952.
4. Hosler, R. M.: A Manual on Cardiac Resuscitation, Springfield, 1954, Charles C Thomas, Publisher.
5. MacQuigg, R. G.: A Transperiosteal Intercostal Incision, Am. Surgeon **20**: 619, 1954.
6. Batelli, F.: Le rétablissment des fonctions du coeur et du systéme nerveux central aprés l'anémia totale, J. physiol. et path. gén. **2**: 443, 1900.
7. Hooker, D. R., Kowenhoven, W. B., and Langworthy, O. R.: The Effects of Alternating Currents on the Heart, Am. J. Physiol. **103**: 444, 1933.
8. Stearns, N. S., Maison, G. L., and Stutzman, J. W.: Cardiac Resuscitation From Induced Ventricular Fibrillation: The Influence of Massage, Procaine, and Electric Shock, Am. J. Physiol. **164**: 601, 1951.
9. Howkins, J., McLaughlin, C. R., and Daniel, P.: Neuronal Damage From Temporary Cardiac Arrest, Lancet **250**: 488, 1946.
10. Tureen, L. L.: Effect of Experimental Temporary Vascular Occlusion on the Spinal Cord, Arch. Neurol. & Psychiat. **35**: 789, 1936.
11. Gomez, L., and Pike, F. H.: The Histological Changes in Nerve Cells Due to Total Temporary Anemia of the Central Nervous System, J. Exper. Med. **11**: 257, 1909.
12. Hoefer, P. F. A., Putnam, T. J., and Gray, M. G.: Experimental "Encephalitis" Produced by Intravenous Injection of Various Coagulants, Arch. Neurol. & Psychiat. **39**: 799, 1938.

SURGICAL PAROTITIS, A VANISHING DISEASE

JOHN R. ROBINSON, M.D., ST. LOUIS, MO.

(From the Department of Surgery, Washington University School of Medicine, and Barnes Hospital)

THIS condition, while not common, was of sufficient frequency and interest to merit an occasional study and review. This is a review of cases of acute parotitis developing after surgical procedures at Barnes Hospital, St. Louis, Missouri, from 1921 through 1952.

The actual incidence of surgical parotitis over the years has been rather difficult to ascertain. Reports concerning this entity up to 1943 indicate that it was an uncommon, but a rather serious, complication in larger medical centers, especially where major gastrointestinal surgery was performed frequently. Interestingly, the incidence of 1 in 2,835 cases at Barnes Hospital from 1921 to 1943 closely parallels that reported by others (Table I).

The pathogenesis of this disease is still obscure. Discussions of the probable methods of infection can be found dating back to Paget[7] (1886). Very little has been added to his original ideas. There are four possible portals through which bacteria may gain entry into the gland: (1) the lymphatics; (2) the blood stream; (3) the parotid duct; and (4) by direct extension from contiguous tissues. There are many arguments for each of these. However, everyone agrees that the dry mouth appears to be an associated, if not a causative, factor. For one reason or another saliva does not flow properly. Saliva contains mucin which has a definite bacteriostatic effect. Presumably, when this ceases to flow, the organisms of the mouth are allowed to ascend the duct without any of the usual deterrents.

Most often the hemolytic *Staphylococcus aureus* was found to be the bacterium in the gland in this series. However, occasionally streptococci and gram-negative rods were also found. When cultures were taken simultaneously from the ducts and the gland, different organisms were often found in the two loci. Vincent's organisms were not found in either ducts or glands at Barnes Hospital, but, at other places, they have been found, although rarely.

Cultures were taken directly from the gland in eleven cases, and from the duct in nineteen cases. Hemolytic *Staphylococcus aureus* was found ten times in the gland and twelve times in the duct; hemolytic streptococci were found twice in the gland and seven times in the duct. The predominance of staphylococci is in accord with the general experience of others. Positive blood cultures were obtained from four patients in this series. In three of these the organism in the blood corresponded with that obtained from the gland. In one instance hemolytic streptococci were grown from the blood while the gland contained hemolytic staphylococci.

Received for publication March 21, 1955.

TABLE I

AUTHOR	NO. CASES	ADMISSIONS	INCIDENCE
Pique[1]	2	7,200	1/3,600
Beckman[2]	3	1,827	1/2,270
Collens[3]	8	6,100	1/720
Gish[4]	44	95,367	1/2,176
Combs[5]	9	13,000	1/1,450
Clifford[6]	10	22,750	1/2,275

In order to test the contention that parotitis is a vanishing disease, this series has been divided into time-periods, corresponding to those in which distinct changes in postoperative surgical management were made. Period I includes the years 1921 through 1927. It is used as the control for the other groups. The second group includes the years 1928 through 1937, and represents a period when much attention was being paid to aseptic intestinal anastomoses, fluid balance, and intestinal decompression by intubation during gastrointestinal surgical procedures. During this era the mortality rates attending colonic and gastric operations were greatly reduced. The third period represents the "sulfa" era and includes the years 1938 through 1942. During this time the sulfonamide drugs were used extensively to treat infection and to prepare the gastrointestinal tract. The fourth period, 1943 through 1952, is the antibiotic era. This decade started with the introduction of penicillin during 1943. Other potent antibiotics were introduced thereafter.

Table II shows what has happened to the incidence of surgical parotitis during these four periods. Table III lists the mortality rates and total number of cases of the disease seen during these times. Figs. 1 and 2 illustrate the data contained in Tables II and III by means of bar graphs. All data in Tables II and III have been compared with the control group I employing chi square. The chi square and probabilities are included in the tables.

TABLE II. INCIDENCE

PERIOD	CASES	TOTAL ADMISSIONS	INCIDENCE	PER 1,000	χ^2	PROBABILITY
1921-1927	8	21,735	1/ 2,718	3.680	x	x
1928-1937	18	51,986	1/ 2,888	3.348	0.015	0.0
1938-1942	4	36,938	1/ 9,248	1.82	6.1	0.01-0.07
1943-1952	2	115,144	1/57,572	0.1738	35.0	0.0001

TABLE III. DEATHS

PERIOD	DEATHS	TOTAL CASES	PER CENT	χ^2	PROBABILITY
1921-1927	7	8	87.6	x	
1928-1937	6	18	33.33	6.5	0.01
1938-1942	1	4	25.0	4.68	<0.05 >0.02
1943-1952	0	2	0.0	5.83	0.01

The frequencies of parotitis in Periods I and II are the same; chance can readily account for the observed difference. This indicates that the advances in pre- and postoperative care that effectually reduced operative mortality during this time exerted no appreciable influence upon the frequency of surgical parotitis (see Discussion).

The comparison of Period III with Period I indicates that the probability that the incidence of the disease in these two series is identical is less than 0.01.

The comparison of Periods I and IV shows that there is about 1 chance in 10,000 that the incidences in these two series could be identical.

The death rates associated with surgical parotitis in Periods I and II are different, although the incidences were not. Chi square comparison of the death rates in these two periods shows that the probability of their being identical is about 1 in 100. That is to say, the lower death rate in Period II is very likely not a chance phenomenon. During Period II (1928 through 1937) the treatment of surgical parotitis differed from that used during Period I. The improvement in mortality rates between Periods I and II of our study is ascribable to radiation therapy (see Discussion).

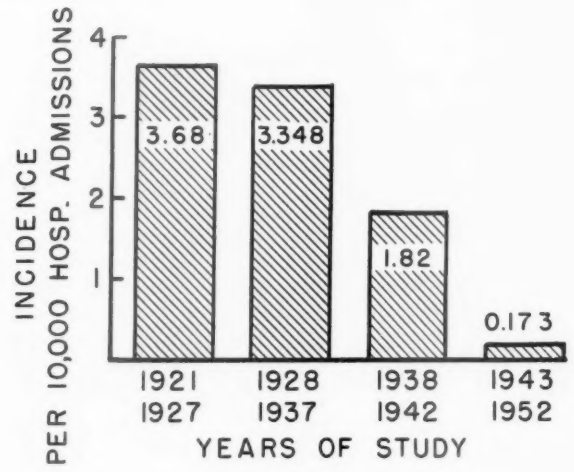


Fig. 1.

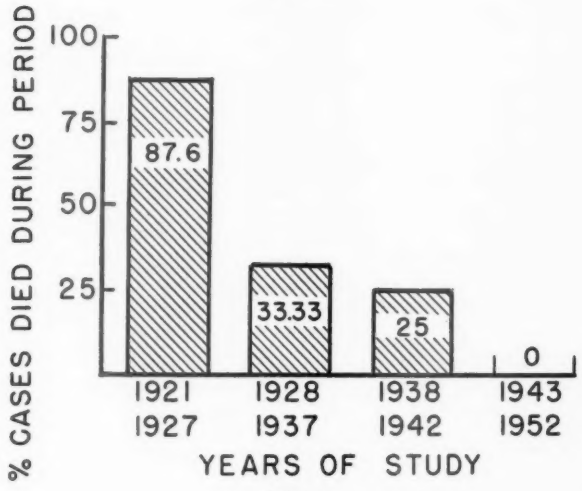


Fig. 2.

Comparison of Period I cases with those of Period III in Table II shows that there are about 3 chances in 100 that the mortality rates are identical. This indicates that the mortality rate during Period III was less than it was in Period I, but probably no different than it was during Period II.

The comparison of the mortality rates during Periods I and IV was handicapped by the few cases available for comparison. However, the probability that the death rates in the two series were identical was less than 1 in 100, and warrants the statement that the mortality from surgical parotitis during Period IV was less than it was during Period I.

DISCUSSION

Surgical parotitis has practically disappeared as a morbid and lethal postoperative complication. The mortality from this entity began to fall before the advent of antibiotics (sulfa, penicillin, and so on), while the incidence did not decline until they were introduced.

During the 1921-1927 period, the treatment of surgical parotitis was chiefly centered about increasing the salivary flow by using various substances such as chewing gum, and so on. The local treatment of the inflamed gland with ice packs or heat also was used. In the late 1920's and early 1930's, Rankin and Palmer,[8] Charlton,[9] and others described the use of radiation therapy to dry up the infected gland; if this was instituted early the results were quite good. The improvement in mortality rate between Periods I and II of our study is ascribable in all likelihood to radiation therapy.

There are probably many reasons why postoperative surgical parotitis has practically vanished, and antibiotic therapy is likely the chief reason for its disappearance. This is certainly supported by the remarkable decrease in incidence coincident with the introduction of sulfonamides (Period III) and penicillin (Period IV) (Fig. 1). Since 1943, it has been the practice of the resident staff at Barnes Hospital to use antibiotics freely postoperatively. This probably accounts for the almost complete disappearance of acute surgical parotitis in this hospital. However, other things, such as better control of fluid and electrolyte balance, better oral hygiene, blood replacement, and good feeding practices, may play some small part in the lowered incidence of this disease. We may in our lifetime be permitted a chance to test just how much value antibiotics really are in preventing this disease. More and more organisms are becoming antibiotic-resistant as a result of indiscriminate use of these drugs. Should this continue, in the future we may expect an antibiotic-resistant staphylococcic organism to give rise again to a rebirth of surgical parotitis.

The net mortality rates did not decline significantly while the sulfas were the only available antibacterial agents (compare Periods II and III of Fig. 2). However, the gross mortality associated with this entity did decline by virtue of the fact that the incidence was reduced during the sulfa era. The mortality rate during the period 1943-1952 was zero. This does not mean that penicillin is completely effective as a therapeutic agent, because only two cases of the disease occurred in this time. However, we can say that penicillin and other antibiotics are most effective preventives.

Of interest is the change in the elapsed time between the operation and the onset of postoperative parotitis between Periods I and IV. The two cases which occurred during the antibiotic era appeared much later than those of prior periods. This change has been observed by many with other postoperative

surgical infections since the advent of antibiotics. In Groups I and II patients (1921 through 1938), the incubation period lasted from five to eight days. The two cases of the antibiotic Period IV had incubation periods of 25 and 35 days, respectively.

Table IV indicates the age at death of the patients succumbing to this disease. We include this merely to dispel an idea held by many, including ourselves, that most patients with parotitis were in their seventh, eighth, or ninth decades of life, and in an extremely debilitated condition. The study seems to indicate that the disease did strike the aged, debilitated patient more readily than others, but is was often seen in young individuals.

TABLE IV. AGE INCIDENCE

GROUP	NO. CASES	NO. DEATHS
0-19	1	1
20-29	3	1
30-39	4	1
40-49	8	1
50-59	8	6
60-69	5	1
70-79	2	2
80-89	1	1

The treatment of surgical parotitis at present seems to be prophylaxis in preventing its development by institution of antibiotic therapy. For those cases already developed, Terramycin seems to be the most effective antibiotic in combating its spread. King[10] described a method of intraductal irrigation with Terramycin and claimed it very effective. When pus is present in the gland, incision and drainage must still be carried out.

SUMMARY

Postoperative surgical parotitis has practically vanished and prophylactic antibiotic therapy is probably the chief reason for its disappearance. Mortality from the disease was definitely lowered by x-ray therapy. The low mortality during the sulfa and antibiotic periods may be due in most part to the low frequency of the disease during these periods.

REFERENCES

1. Pique, L., and Toubert, J.: Bull. et mém. Soc. nat. de chir. de Paris **29**: 709, 1903.
2. Beckman, E. H.: Proc. Staff Meet., Mayo Clin. (papers of St. Mary's Hosp.), vol. V., Philadelphia, 1913, W. B. Saunders Company, p. 776.
3. Collens, C. V.: Surg., Gynec. & Obst. **28**: 404, 1919.
4. Gish, E. R., and Caughlin, W. T.: Arch. Surg. **45**: 361-405, 1942.
5. Combs, C. N.: Anesth. & Analg. **7**: 92, 1928.
6. Clifford, W. J.: South. Surgeon **16**: 584-593, 1950.
7. Paget, S.: Lancet **1**: 86, 1886.
8. Rankin, F. W., and Palmer, B. M.: Ann. Surg. **93**: 837-843, 1931.
9. Charlton, P. H.: Ann. Surg. **93**: 837, 1931.
10. King, G. D.: Lahey Clin. Bull. **7**: 244-250, 1950.
11. Moyer, C. A.: Personal communication.

PERCUTANEOUS SPLENIC PORTOGRAMS IN DOGS: TECHNIQUE AND EXAMPLES OF USEFULNESS

ROBERT E. DAVIS, M.D.,* GEORGE A. HALLENBECK, M.D., FRANK R. LICHTENHELD, M.D.,* AND JOHN H. GRINDLAY, M.D., ROCHESTER, MINN.

(From the Sections of Surgery and Surgical Research, Mayo Foundation† and Mayo Clinic)

WITH the demonstration by Abeatici and Campi[1] in 1951 that contrast media injected into the spleen were projected directly into the portal circulation, this venous system for the first time became available to roentgenologic visualization without the necessity of surgical exposure. The use of direct splenic (percutaneous) venography in man has now been reported by many authors in conjunction with surgical treatment of portal hypertension. It is our present purpose to describe a technique by which the portal circulation of dogs can be made almost as readily available for injection as is the systemic venous system.

It is considerably more difficult to inject percutaneously into the slender mobile spleen of the dog than is the case with the human spleen.[2] Abeatici and Campi reported use of splenic portograms in dogs, but did not state whether or not surgical exposure was used. Dreyer[3] has performed a subcutaneous explantation of the entire spleen, in which the abdominal wall is closed around the splenic vessels. We wish to describe a surgical method in which the dog's spleen is partially explanted into subcutaneous tissues and fixed, becoming permanently accessible for direct injection, without alteration of position or vascular relationships of the major portion of the spleen.

METHOD

With the dog under endotracheal ether anesthesia and with sterile technique, a midline epigastric incision is made. The areolar space beneath the panniculus carnosis on the left side of the abdomen is entered with one or two scissors' cuts. This pocket can then be readily enlarged by blunt dissection with the fingers, no bleeding being encountered. By palpating the mid-point of the spleen as it lies in its normal position within the abdomen, a site is selected for explantation of the lower pole near the lowest portion of the left costal margin. At this point, through the subcutaneous pocket, a transverse or gridiron incision is made, as long as the spleen is wide, through all musculofascial layers of the abdominal wall. The inferior tip of the spleen is then pulled through this incision to lie in the subcutaneous pocket with practically no distortion of its normal position and configuration. One suture is placed between the mesentery adjoining the splenic capsule and the adjacent fascia, to resist the tendency of respiratory movement to retract the spleen into the abdominal cavity. The midline incision is then closed in a routine manner.

Much variation is possible in the technique of injection. Our studies have usually been performed with the animal in the prone position, under ether anesthesia. The supine or lateral position is equally feasible. We have thought that the enlargement of the spleen accompanying the use of barbiturates is a contraindication to the use of this type of

Received for publication March 12. 1955.

*Fellow in Surgery.

†The Mayo Foundation is a part of the Graduate School of the University of Minnesota.

anesthesia. Dogs can readily be trained to cooperate in the procedure without anesthesia and this has been done in many cases. A needle of 18 to 20 gauge is introduced into the center of the subcutaneous portion of the spleen, its location verified by the aspiration of blood, and the injection of contrast medium is made during a five-second period at the end of which the roentgen exposure is made. In a dog weighing 7 kilograms, 10 ml. of 75 per cent sodium iodomethamate (Neo-Iopax) gives good visualization of the extrahepatic and intrahepatic portal system. In dogs prepared with Eck fistulas, it has been necessary to use 20 ml. of contrast medium. In order to demonstrate possible uses of the method, dogs have also been prepared with Eck fistulas, strictures of the portal vein, and ligatures of the portal vein.

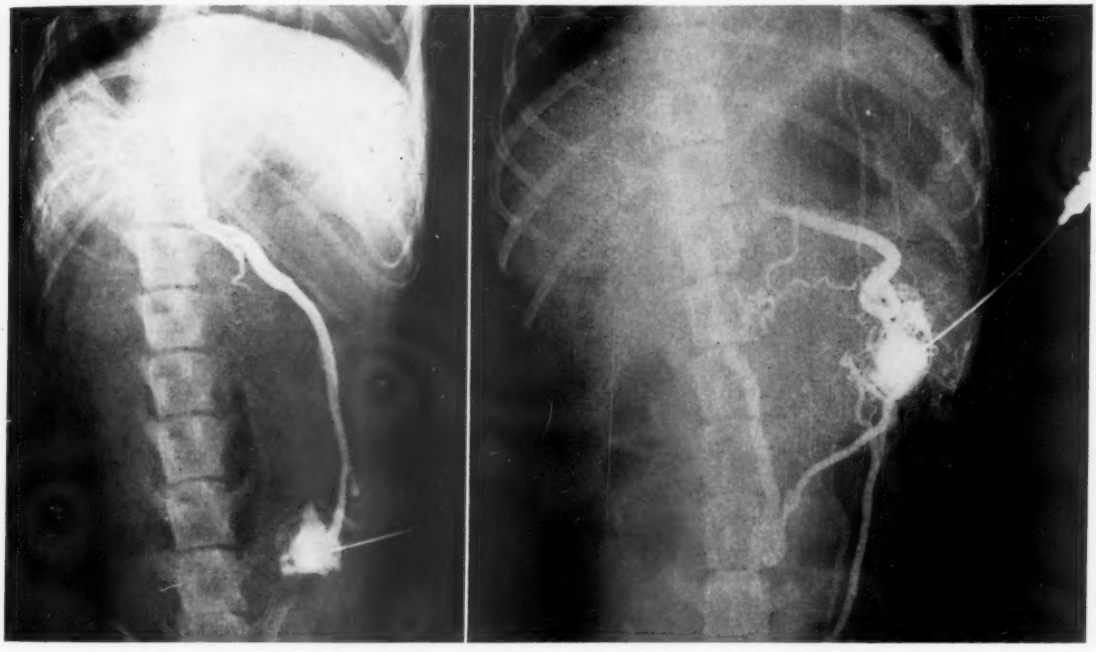


Fig. 1. Fig. 2.

Fig. 1.—Normal splenic portogram showing "streamline" filling defect in terminal portion of splenic vein and first portion of portal vein.

Fig. 2.—Strictured portal vein with tortuosity of splenic vein and retrograde and collateral flows.

RESULTS

Eight dogs underwent subcutaneous fixation of the spleen as described. Of these, one died of causes unrelated to this procedure, and two developed the Eck fistula syndrome and died five and eight weeks, respectively, after production of the Eck fistulas. One spleen retracted into the abdominal cavity immediately after operation, and one other retracted between two and eight weeks after operation. This difficulty has not occurred since we have realized the necessity of placing the fixation suture in the splenic mesentery immediately adjacent to the splenic capsule.

Visualization of the splenic vein, portal vein, and intrahepatic portal radicles has been routinely good (Figs. 1 and 2) except when the needle tip has inadvertently been placed outside the substance of the spleen, or on repeat injections when the needle is introduced into the small subcapsular hematoma

that is occasionally seen after previous injections. Repeat injections are feasible at intervals of seven to fourteen days. We have performed splenic venography as long as thirty-six weeks after preparation, and believe that the spleen remains available permanently.

In dogs prepared with Eck fistulas for other purposes, splenic portograms have been useful for determining the patency of the portacaval anastomoses at any time during their course (Fig. 3). Because of the hazard of anesthesia to these dogs, the studies have usually been made without anesthesia.

Fig. 3.—Eck fistula with outline of patent portion of portal vein and inferior vena cava.

Visualization of portal systems after ligation or the causation of stricture has consistently demonstrated increased tortuosity of the splenic vein, retrograde flow into the mesenteric veins, or collateral circulation cephalad through veins in the region of the esophagus. A few collateral vessels to the body wall (usually the internal mammary veins) are frequently seen after subcutaneous fixation of the spleen, and these seem to be increased in the few cases in which vascular adhesions to the subcutaneous tissues have been produced by through-and-through mattress sutures placed in the spleen to anchor it to the body wall.

Another use of the explanted spleen has been demonstrated in studies in other investigations in which it has been possible to administer infusions of Glucagon regularly into the portal venous system.

We have been surprised to find little evidence of "streamlining" of the portal circulation, as have other investigators.[4-8] In reviewing the evidence for streamlining, we have come to the conclusion that the present method temporarily alters the portal pressure and flow relationships, momentarily blocking the emergence of blood from the mesenteric veins.[9, 10] An occasional film has been suggestive of streamlining in the first centimeter of the portal vein, but

the contrast medium has always been uniformly distributed into all intrahepatic portal radicles. Dreyer and Budtz-Olsen[3, 5] also commented on the small filling defect sometimes seen at the entrance of the superior mesenteric vein.

SUMMARY

The method of subcutaneous fixation of the spleen is a useful procedure in animal research in which it is desired to inject substances into the portal venous system. Splenic portography performed through the exteriorized spleen gives reliable information on the topography and functional status of the portal and splenic veins. Such studies may be repeated at frequent intervals without the necessity of surgical exposure and deep anesthesia.

REFERENCES

1. Abeatici, S., and Campi, L.: La visualizzazione radiologica della porta per via splenica, Minerva med. **42**: 593-594, 1951.
2. Tori, Giulo, and Scott, W. G.: Improved Method for Splenoportography Using Biplane Serialized Exposures, Am. J. Roentgenol. **70**: 237-241, 1953.
3. Dreyer, B.: Splenic and Portal Venography, Quart. J. Exper. Physiol. **39**: 93-100, 1954.
4. Moore, G. E., and Bridenbaugh, R. B.: Roentgen Demonstration of the Venous Circulation in the Liver: Portal Venography, Radiology **57**: 685-689, 1951.
5. Dreyer, B., and Budtz-Olsen, O. E.: Splenic Venography: Demonstration of the Portal Circulation With Diodone, Lancet **1**: 530-531, 1952.
6. Gvozdanović, V., Hauptmann, E., Najman, E., and Oberhofer, B.: Percutaneous Splenic Venography, Acta radiol. **40**: 17-26, 1953.
7. Bahnson, H. T., Sloan, R. D., and Blalock, Alfred: Splenic-portal Venography: A Technique Utilizing Percutaneous Injection of Radiopaque Material Into the Spleen, Bull. Johns Hopkins Hosp. **92**: 331-345, 1953.
8. Du Boulay, G. H., and Green, B.: Portal Venography in Banti's Disease, Brit. J. Radiol. **27**: 423-434, 1954.
9. Copher, G. H., and Dick, B. M.: "Stream Line" Phenomena in the Portal Vein and the Selective Distribution of Portal Blood in the Liver, Arch. Surg. **17**: 408-419, 1928.
10. Hahn, P. F., Donald, W. D., and Grier, R. C., Jr.: The Physiological Bilaterality of the Portal Circulation: Streamline Flow of Blood Into the Liver as Shown by Radioactive Phosphorus, Am. J. Physiol. **143**: 105-107, 1945.

SURGICAL TREATMENT OF TRANSPOSITION OF THE GREAT VESSELS

EARLE B. KAY, M.D., AND FREDERICK S. CROSS, M.D., CLEVELAND, OHIO

(From the Medical and Surgical Services of St. Luke's and St. Vincent Charity Hospitals and also from the Surgical Laboratory of St. Luke's Hospital)

COMPLETE transposition of the aorta and pulmonary artery is a common form of congenital heart disease for which, as yet, there is no successful operative technique. It occurs with about the same frequency as tetralogy of Fallot. Approximately 25 per cent of the infants with this condition die within the first month of life and 75 per cent within the first year. The pulmonary and systemic circulations are essentially separate circulations except for a certain degree of intermixing of the blood between the two sides by means of septal defects or patency of the ductus arteriosus. Not infrequently the associated defect present which allows for the intermixing of the blood between the two circulations may be insignificant. In approximately one-third of 29 cases reported by Lillehei and Varco[1] a patent foramen ovale, with or without a persistent patent ductus arteriosus, was the only other associated cardiac anomaly.

In patients with this condition, the aorta arises anteriorly from the right ventricle and the pulmonary artery posteriorly from the left ventricle. These infants are more cyanotic, dyspneic, and feeble than infants with tetralogy of Fallot. The heart size may be normal at birth but soon becomes enlarged. The shadow cast by the great vessels at the base of the heart, as noted in an anteroposterior roentgenogram, is narrower than noted in other forms of cyanotic heart disease. There is frequently vascular congestion of the lung fields. Pressures taken at the time of operative procedures, as well as preoperatively, show the pressure within the pulmonary circulation to be almost as high as the systemic circulation. Systolic murmurs are usually present but are not characteristic. Cardiac catheterization and angiocardiograms are useful in making a positive diagnosis. However, a presumptive diagnosis can usually be made on clinical findings alone.

Hanlon and Blalock[2] in 1948 performed various shunt operations and found that the best results were obtained when an atrial septal defect was created in combination with an anastomosis between the subclavian and pulmonary arteries. These patients were only moderately improved, and their arterial oxygen saturation generally remained below 75 per cent. Lillehei and Varco[1] in 1953 reported on the treatment of transposition of the great vessels by directing their efforts to transposing the venous return. Two patients were improved by anastomosing the right pulmonary veins to the right atrium.

Supported in part by Research Grant from the Cleveland Area Heart Society.
Received for publication April 18, 1955.

In four instances the inferior vena cava was anastomosed to the left atrium at the site of the cardiac end of the transected right pulmonary veins without success. Mustard and associates[3] in 1954 reported on the use of an extracorporeal circulation employing a pump and monkey donor lungs for oxygenation in which the aorta with the left coronary artery and the pulmonary artery were retransposed. This theoretically is the ideal type of surgical correction but as yet has not met with any success. In the same year, we reported a cross-shunting technique by which the aortic and pulmonary valves could be by-passed by means of valved plastic shunts between the ventricles and aortic vessels.[4] There were no successes in the three clinical cases in which this technique was employed. During the same year Bjork and Bouckaert[5] employed homologous arterial grafts experimentally to crisscross the circulation in dogs.

During the past year we have employed a new technique that appears promising even though we have not been successful in the two patients upon whom it has been performed. One infant was 4 weeks of age and the other 3½ months. The 4-week-old child was the second one, performed in December, 1954. Its femoral arterial saturation was only 18 per cent. An oxygenated transfusion was employed during the procedure. The use of oxygenated blood for such patients is undoubtedly beneficial.

The technique employed in these two infants consisted of the following: (Fig. 1) A transsternal approach through the third intercostal space was used. In one instance, the right pulmonary artery was first anastomosed to the aorta. In the other, the pulmonary conus was anastomosed first to the arch of the aorta just distal to the innominate artery. The sequence of the anastomoses is probably not important. The following description will pertain to just one sequence, in which the right pulmonary artery is transected at the conus. The distal end is anastomosed to the side of the aorta. This anastomosis is then opened so that blood from the right ventricle can circulate to both the right lung and to the arch of the aorta. During the performance of this anastomosis, oxygenation is provided by the left lung. The proximal end of the pulmonary artery is closed flush with the pulmonary conus. The end of the pulmonary conus is then anastomosed to the arch of the aorta, following which this anastomosis is opened. The aorta is transected proximal to the innominate artery and the two ends are closed. The left pulmonary artery is transected close to the pulmonary conus. A gap between the distal end of the left pulmonary artery and aorta results, which is bridged by a homologous arterial graft. The proximal end is closed. This sequence of anastomoses results in complete retransposition of the aorta and pulmonary artery.

The color of both of the infants was markedly improved following operation. The color of the first infant, 3½ months old, operated upon in November, 1954, appeared entirely normal. He responded immediately following operation and appeared to be doing entirely satisfactorily for five hours postoperatively, when he suddenly expired. The autopsy failed to reveal the

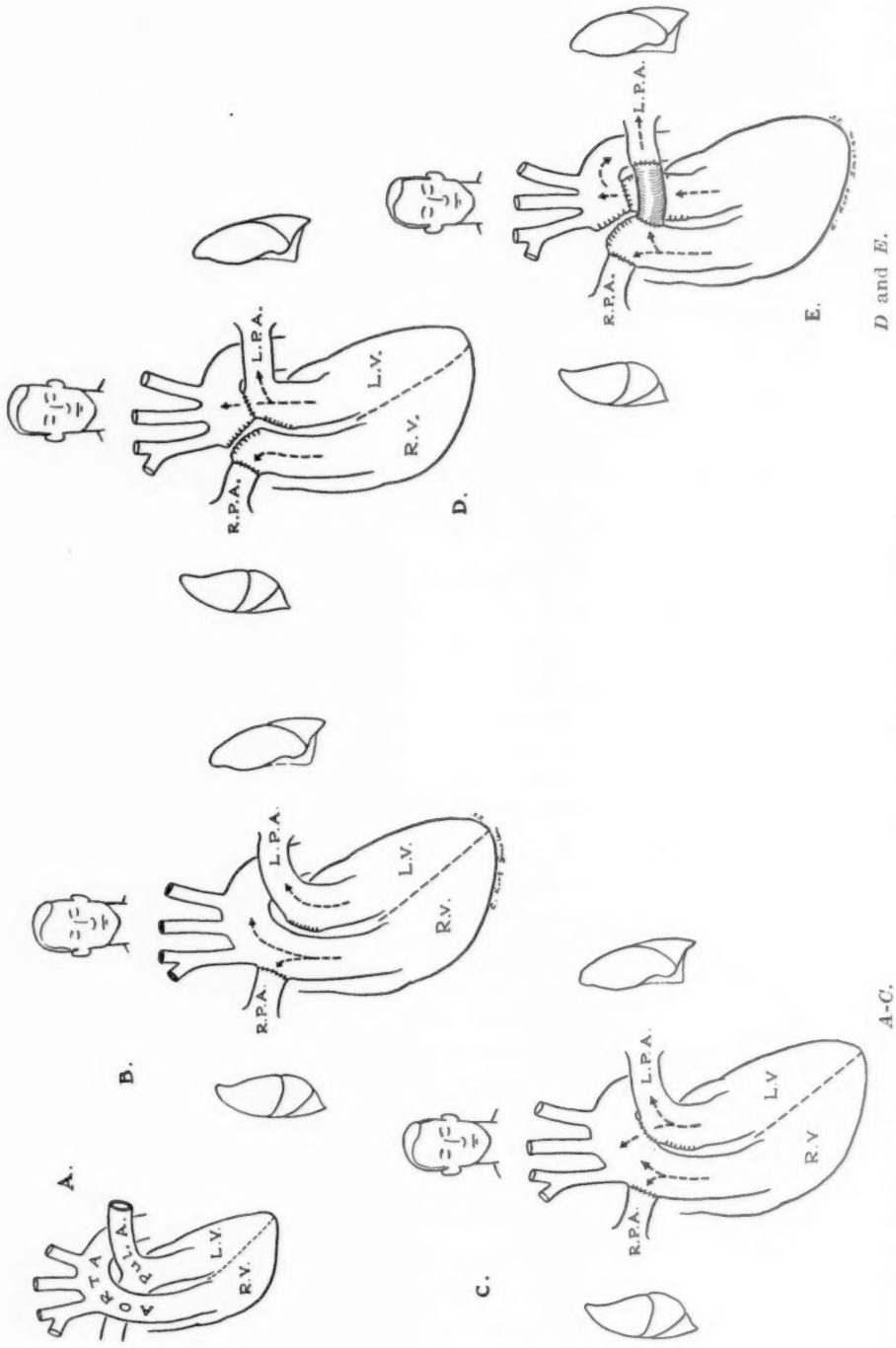


Fig. 1.—See text for discussion.

A-C. *D* and *E.*

cause of death. The second infant went through the operative procedure most satisfactorily. The color appeared very good immediately afterward for about five to ten minutes. It then again became cyanotic and respirations were feeble. The infant was thought to have a cardiac arrest. Consequently, the chest was immediately opened, whereupon it was noted that the rigid homologous graft had kinked and occluded the pulmonary artery during closure of the chest incision. The graft used was a trifle too long. During the operative procedure with the chest incision open, it appeared satisfactory. However, with closing the incision the graft appeared to have kinked, occluding the outflow from the right ventricle and aorta at the site of the anastomosis. This is a technical error which can be avoided in the future.

Only experience will decide how necessary it is to transplant one or both coronary arteries. When one considers that the preoperative oxygen saturation of the coronary vessles in patients with transposition of the arteries ranges between 20 to 40 per cent, then improvement should follow if the venous blood is increased to about 75 per cent. However, because of the possible necessity of transposing the coronary arteries and particularly the left coronary artery in order to provide sufficient oxygenation of the myocardium, we are attempting to solve this problem in the laboratory by arteriolizing the left coronary artery by anastomosing it to the left subclavian artery. In our opinion it is unwise to occlude temporarily the coronary arteries in patients with transposition during any attempt to transplant them because of the danger of initiating irreversible ventricular fibrillation. It is questionable at this time whether such an anastomosis should be performed at the same time as the other anastomoses or at a later date. The present opinion is that it should be deferred to a later date.

SUMMARY

Transposition of the aorta and pulmonary artery is discussed. The historical developments in the surgical correction of this condition is presented, as well as a new technique that appears to have merit.

ADDENDUM

Since this report was written a third infant, 16 months of age, has been operated upon by this technique. In this instance a nylon tube impregnated with silicone[6] was employed to bridge the gap between the new pulmonary conus and the distal end of the left pulmonary artery. The infant succumbed three hours after completing the retransposition of the vessels from progressive pulmonary congestion and hemorrhage, during which time there was an opportunity to make several significant observations. This pulmonary congestion and the diffuse petechial interstitial hemorrhages were also noted in the other two patients, but their significance was not recognized. By means of the strain-gauge manometer, pressure recordings were obtained to provide a better understanding of the underlying cardiac dynamics. The pressure within the right ventricle was 110 mm. Hg, pulmonary conus 90 mm. Hg, left ventricle 90 mm. Hg, and the arch of the aorta 60 mm. Hg. It was felt that the increasing pulmonary congestion and hemorrhage probably resulted from the inability of the high-resistance bed of the pulmonary circulation to further adapt itself acutely to the increased pressure from the hypertrophied right ventricle. An attempt was made to reduce the pulmonary blood flow by various methods, including the reduction

in size of the pulmonary conus by means of a constricting tape. It was necessary to constrict the pumonary conus from 2 cm. to approximately 0.5 cm. in diameter before pressures of 60 mm. Hg in the pulmonary artery distal to the point of partial obstruction and 96 mm. Hg in the right ventricle were obtained.

It was necessary to constrict almost completely the pulmonary conus before a normal pressure within the pulmonary artery was obtained. This degree of obstruction was thought to be detrimental. After three hours of attempting various maneuvers, the infant died. Post-mortem examination revealed a moderately large ventricular septal defect as was noted in the other two patients. In view of this experience, it may not be possible to transpose completely the arterial trunks in patients with transposition of the great vessels.

REFERENCES

1. Lillehei, C. W., and Varco, R. L.: Certain Physiologic, Pathologic, and Surgical Features of Complete Transposition of the Great Vessels, SURGERY 34: 376, 1953.
2. Hanlon, C. R., and Blalock, A.: Complete Transposition of the Aorta and Pulmonary Artery. Experimental Observations on Venous Shunts as Corrective Procedures, Ann. Surg. **127**: 385, 1948.
3. Mustard, W. T., Chute, A. L., Keith, J. D., Sirek, A., Rowe, R. D., and Vlad, P.: A Surgical Approach to Transposition of the Great Vessels With Extracorporeal Circuit, SURGERY **36**: 39, 1954.
4. Cross, F. S., Kay, E. B., Jones, R. D.: A Simple Shunting Technique for Surgery of the Aortic and Pulmonary Valves and Proximal Great Vessels, J. Thoracic Surg. 28: 229, 1954.
5. Bjork, V. O., and Bouckaert, L.: Complete Transposition of the Aorta and Pulmonary Artery, J. Thoracic Surg. **28**: 632, 1954.
6. Hudack, Stephen S., Whittlesey, Robert H., and Jones, James C.: Personal communication (with reference to the use of plastic materials as arterial substitutes).

A NEW TWO-LEVER COMPRESSION CLAMP FOR VASCULAR SURGERY

A. M. Dogliotti, M.D.,* and G. Guglielmini, M.D.,** Turin, Italy

(From the University of Turin)

THREE years ago we described a special type of clamp—a three-branched clamp—with which it was possible to simplify the execution of a correct vascular anastomosis.

This tool, devised at first to perform the aorta-pulmonary anastomosis according to Potts' method, proved thereafter to be very useful for all vascular anastomoses—either arterioarterial or arteriovenous—to be performed in any part of the body (splenorenal, portacaval, splenosplenic, carotid-jugular, and so on).

The advantages of the clamp we dealt with at that time may be schematically illustrated as follows: (a) It allows one to maintain the blood circulation inside the vessels all the time during the operation. (b) It is easily placed and easily removed. (c) The vessel walls to be sutured are held motionless at the same level. (d) It is possible to avoid the all-round isolation of the vessels from their collateral branches; this is especially useful in cases of isolation of the aorta.

Recently we introduced some important modifications with the previously described instrument (Fig. 1).

The more important modifications are: (1) Alteration of the shape in order to allow a better deep fitting of the instrument. (2) A mechanical device with a lever for maneuvering and controlling the working branches of the clamp. These are put into action by means of small wheels placed at a distance on the handle of the tool, so that they work very smoothly on opening and closing, while they exert a powerful pressure as well.

Owing to the long arms of the levers, the closing strength is a good deal higher than that obtainable with other types of clamps and it secures at the utmost a firm hold of the vessel walls, even in cases of high pressure vessels (aorta).

Furthermore, the two-lever device assists in giving a proper shape to the instrument and in keeping the controlling device—unavoidably cumbersome—as far as possible from the working end, so that the handle of the instrument remains outside and at a distance from the operative field.

As a conclusion, we may say that the clamp we are illustrating here, made according to our latest model, in addition to the general advantages of the former type, shows the following important improvements: (a) doubled closing strength of the branches; (b) improved adjustability in deep operative fields; (c) action which is parallel to and, therefore, concordant with the surfaces involved in the anastomosis; and (d) it ensures a far greater firmness to the operative field.

Received for publication March 15, 1955.

*Professor of Surgery and Director of the Surgical Clinic.

**Assistant Professor of Surgery.

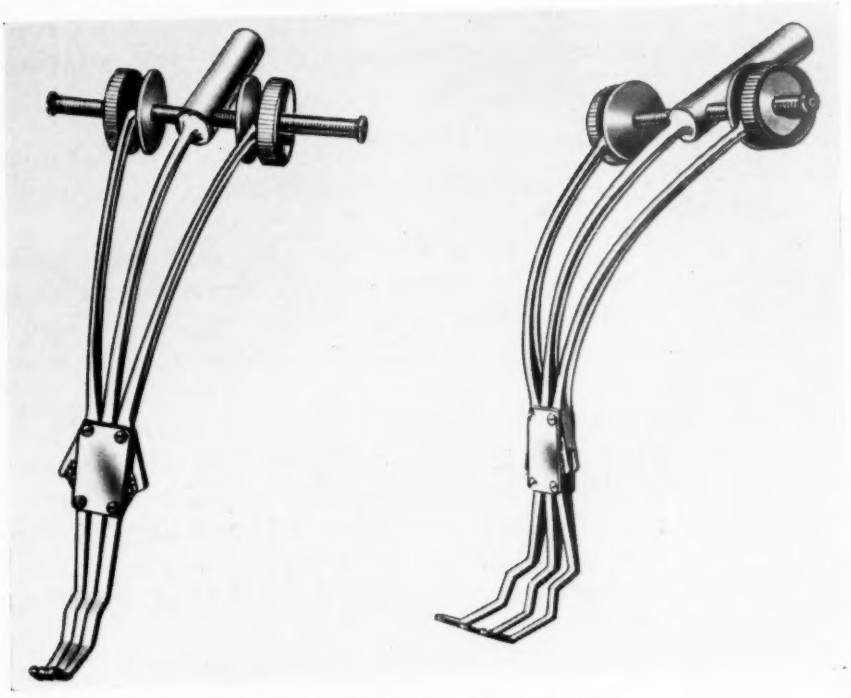


Fig. 1.—Three-branched clamp.

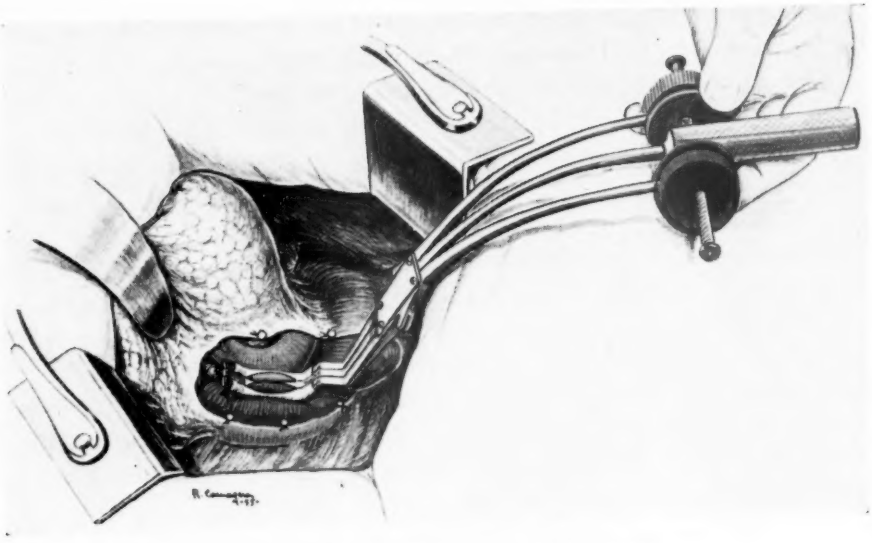


Fig. 2.—Aortic-pulmonary anastomosis.

At present we are making use of it in every case of vascular anastomosis (Blalock's or Potts' operations, portacaval or splenorenal anastomosis), and our already wide experience makes us sure that this type of clamp is a great deal better than all other types utilized thus far.

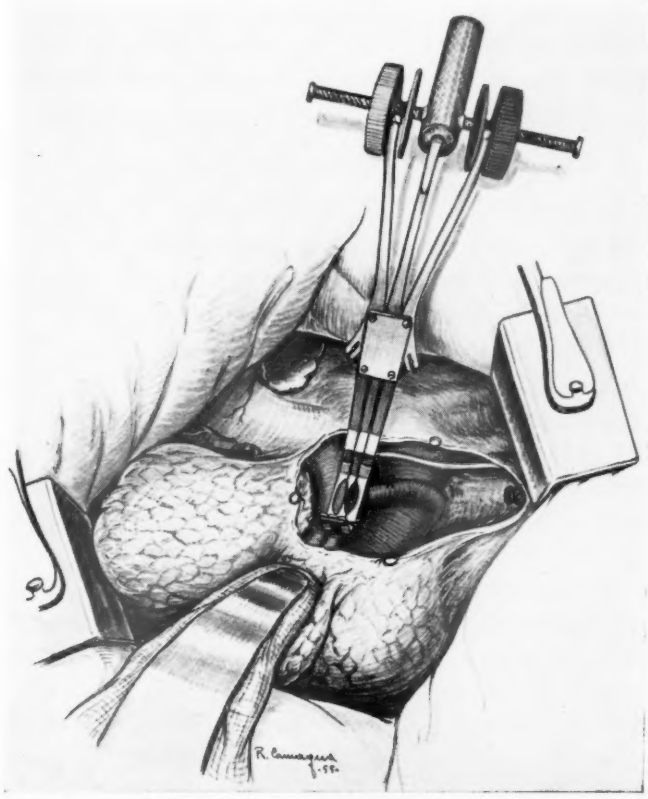


Fig. 3.—Subclavian-pulmonary anastomosis (on the left).

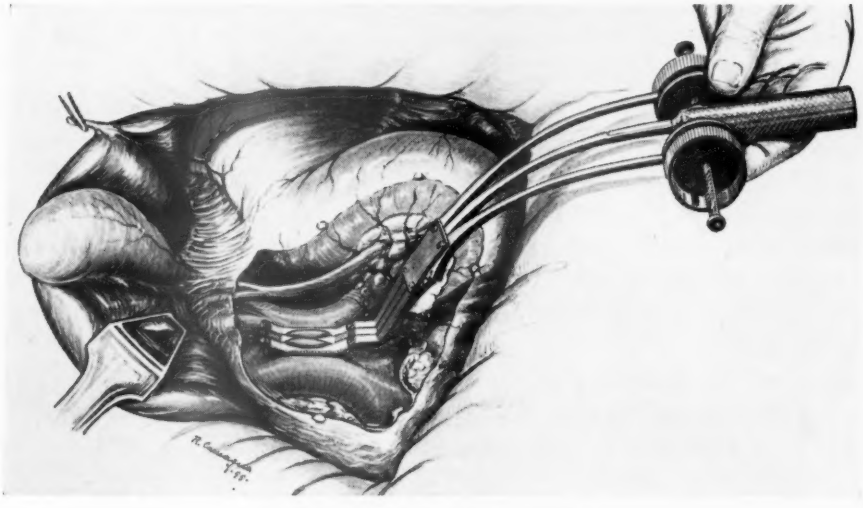


Fig. 4.—Portacaval anastomosis.

FAT EMBOLISM

The Failure of Lipemia to Potentiate the Degree of Fat Embolism Accompanying Fractures of the Femur in Rabbits

Leonard F. Peltier, M.D., Ph.D., Minneapolis, Minn.

(From the Division of Orthopedic Surgery, University of Minnesota Hospitals)

FAT embolism is a condition in which fat appears in the blood stream, not in the form of the chylomicrons of a physiologic lipemia, but in larger aggregates, ranging in size from 10 to 100 microns in diameter. As these particles occlude portions of the vascular bed, especially the lung, local disturbances are produced which, if sufficiently numerous, may eventuate in the death of the patient. Fat embolism is the cause of death in at least one-fourth to one-third of all patients dying after fracture.[5, 7]

The source of this embolic fat has been debated for years. One school of thought believes that the fat reaches the general circulation from the local site of injury; the other believes that it can be mobilized from the body fat depots, especially from that normally carried in the blood stream. In support of the first hypothesis are the following observations: (1) fat embolism is associated most frequently with trauma to the skeleton, and especially to those bones containing fatty marrow; (2) bone marrow embolism from the local site of injury accompanies fat embolism in about 5 per cent of the cases;[4] and (3) when a tourniquet is used proximal to the point of fracture, no fat emboli can be detected in the general circulation until after the tourniquet has been removed and circulation through the injured limb re-established.[3] In support of the second hypothesis are the observations that (1) fat emboli can be produced in the lungs of lipemic animals by the administration of ether by intravenous injection or by inhalation,[1] and (2) there is sufficient readily available fat in the body to provide fat emboli.

It has been known for years that patients with lipemia associated with diseases such as diabetes will exhibit embolic fat in the lung at autopsy. Fat emboli seen under such circumstances, however, appear to be the result of the protective function of the lung in sequestrating unemulsified and unstabilized fat droplets, and should not be confused with embolic fat accompanying skeletal injury, the so-called "traumatic lipemia." Unfortunately, it is impossible to differentiate between embolic fat particles of these two types by histologic means.

In spite of the fact that it has been considered possible that fat emboli associated with skeletal trauma could arise from the general body fat, no one has ever demonstrated a relationship between the incidence of fat embolism

The project was supported by a grant from the Graduate School of the University of Minnesota.

Received for publication May 2, 1955.

following fracture and lipemia, either alimentary or associated with some coincidental disease. The following experiments were carried out in an effort to determine whether or not the presence of a lipemia would potentiate the degree of fat embolism normally associated with fractures in nonlipemic subjects.

METHODS

Adult rabbits weighing 2 to 3 kilograms were anesthetized with pentobarbital sodium administered intravenously. The period of anesthesia in all animals lasted 2½ hours. At the end of this time the animals were sacrificed by a second large dose of pentobarbital sodium. Fractures of the femur were produced in one-half of these animals immediately following induction of the anesthesia. This was carried out by crushing the shaft with a heavy pliers. The fractures were closed and comminuted in type, with minimal displacement.

Lipemia was produced in one group of rabbits by the administration over a period of three weeks of a special diet consisting of pellets with the following ingredients: 985 lbs. dehydrated alfalfa meal, 15 lbs. stabilized fat, 8.2 lbs. cholesterol, and 4.8 lbs. toasted soy flour. No other food was offered to these animals. At the end of twenty-one days, the serum of all of these animals showed a pronounced lipemia. The other group of rabbits was fed the standard laboratory rabbit pellets. None of these animals became lipemic.

Four experiments were carried out. In the first group were normal rabbits without fractures; the second group, lipemic rabbits without fractures; the third group, normal rabbits with fractures of the femur; and the fourth group, lipemic rabbits with fractures of the femur.

At the end of the experiments the rabbits were autopsied, and the lung fixed in 10 per cent Formalin. Four blocks were cut from each of the lungs. Frozen sections from these blocks were cut and stained with Phosphin 3R.[2] They were then examined for fat using fluorescence microscopy. The number of fat emboli in each section were counted. The results were tabulated as follows: 1 to 5 emboli per section +; 5 to 10 emboli per section, ++; 10 to 20 emboli per section, +++; and more than 20 emboli per section, ++++.

RESULTS

The results of these experiments are shown in Table I.

TABLE I. THE INCIDENCE AND DEGREE OF FAT EMBOLISM ACCOMPANYING FRACTURES OF THE FEMUR IN NORMAL AND LIPEMIC RABBITS

	NO. OF RABBITS	NO. $\bar{c}$ FAT EMBOLISM	DEGREE OF FAT EMBOLISM			
			+	++	+++	++++
Normal rabbits without fracture	9	3	3			
Lipemic rabbits without fracture	10	6	5	1		
Normal rabbits with fracture	10	10	3	6	1	
Lipemic rabbits with fracture	12	10	4	3	2	1

Some fat emboli are always present in the uninjured animals. The incidence and degree of this "physiologic" embolism is increased when lipemia is present. The fracture of the femur produced an increase in the incidence and degree of fat embolization in both groups of animals. Although the emboli in the fractured lipemic animals are more numerous than those of the fractured normal group, the total includes the increment of "physiologic" emboli contributed by the lipemia. There is no evidence to indicate that the amount of embolization due to the fracture itself differs significantly.

DISCUSSION

Swank and Dugger,[6] in 1954, reported their results in a study very similar to this. Although they used "fattened" rabbits and "lean" rabbits rather than lipemic rabbits and alipemic rabbits, the procedures were otherwise quite similar, with one exception. There is no control data indicating the degree of fat embolization occurring in the fat rabbit without fracture. For this reason, it is difficult to accept their conclusion that the circulating blood fat is an important source of fat emboli in animals with fractures of the femur. Such a conclusion is not supported by the data presented here.

There are two sources of error in both of these studies. The major difficulty is that of determining the amount of fat embolization which has occurred. The study of frozen sections provides qualitative information easily, but quantitative figures are very inaccurate and are at best merely approximations. A more accurate method of determining the amount of embolic fat present in the lung is sorely needed. The other source of error lies in the amount of depot fat present in skeleton or specifically in the femora of the rabbits. A "lean" rabbit, or one on a normal diet, will increase in weight as it is fattened or placed on a diet which results in the appearance of lipemia. A portion of the retained fat is stored in the fatty marrow. In such a case, the amount of fat in the local site of fracture is greater in the "fat" group, than in the "lean." This discrepancy may be sufficient to explain the difference in the behavior of these two groups without invoking the participation of the general body fat store at all.

CONCLUSION

Fracture of the femur in lipemic rabbits does not produce a significantly greater degree of fat embolism than occurs following similar fractures in alipemic rabbits. This observation supports the conclusion that the source of the embolic fat associated with skeletal trauma is the bone fat at the site of fracture.

REFERENCES

1. Lehman, Edwin P., and Moore, Robert M.: Fat Embolism Including Experimental Production Without Trauma, Arch. Surg. **14**: 621-662, 1927.
2. Peltier, L. F.: The Demonstration of Fat Emboli in Tissue Sections Using Phosphin 3R, a Water-soluble Fluorochrome, J. Lab. & Clin. Med. **43**: 321-323, 1954.
3. Peltier, L. F.: Fat Embolism: The Prophylactic Value of a Tourniquet. In press.
4. Rappaport, Henry, Raum, M., and Horrell, J. B.: Bone Marrow Embolism, Am. J. Path. **27**: 407-426, 1951.
5. Saikku, L. A.: Fat Embolism in Connection With the Treatment of Fractures, Acta chir. scandinav. **108**: 275-282, 1954.
6. Swank, Roy L., and Dugger, Gordon S.: Fat Embolism: A Clinical and Experimental Study of the Mechanisms Involved, Surg., Gynec. & Obst. **98**: 641-652, 1954.
7. Watson-Jones, Reginald: Fractures and Joint Injuries, ed. 4, vol. I, Baltimore, 1952, The Williams & Wilkins Company.

Case Reports

UPPER ESOPHAGEAL RESECTION AND REANASTOMOSIS TO PHARYNX

WILSON WEISEL, M.D., J. J. PINK, M.D., AND J. J. FITZSIMMONS, M.D.,
MILWAUKEE, WIS.

(From the Departments of Surgery and Medicine of Columbia Hospital and the Marquette University School of Medicine)

LIMITED resection of the esophagus and reanastomosis has been proved by experimental[1, 3, 4, 9, 10, 12, 13, 16, 17, 18, 21, 22] and clinical studies[2, 5-8, 11, 14, 15, 19, 20, 23, 24] to be a feasible procedure. The technical surgical precepts which have governed the successful accomplishment of this operation have been careful dissection, recognition and preservation of the segmental blood supply, avoidance of injury to the esophagus by manipulation or application of destructive drugs or substances, the removal of devitalized tissue present after resection, the utilization of small interrupted nonabsorbable sutures for the anastomosis, and the prevention of undue tension at the anastomotic site.

The indications for operations of this type have been primarily confined to palliative resections for malignant lesions or for the removal of benign lesions. The incidence of lesions suitable for resection has apparently been rare, and this fact, as well as the unusual clinical features, has occasioned this report of the following patient.

CASE REPORT

W. P., a 75-year-old white woman, was admitted to the Hospital on June 10, 1953, with chills, fever, and the inability to swallow of four days' duration. The patient gave a history of progressive dysphagia and productive cough during the previous five years, and she was known to have a pharyngoesophageal diverticulum, although because of religious tenets she had delayed considering corrective surgery.

Physical examination showed an acutely ill, toxic, and dehydrated woman. The temperature was 103° F., the pulse rate was 126 and irregular, and auricular fibrillation was present intermittently. Respirations were moderately labored and numbered 26 per minute. There were tenderness and a sense of fullness in the right neck below the thyroid cartilage. Moist and coarse râles were heard posteriorly over the lung bases. There was a Grade 2 pitting edema of the legs. The patient weighed 100 pounds. She was unable to swallow even water, and the attempt to ingest liquids caused choking and immediate regurgitation.

The pertinent findings on clinical examination were elevation of the white blood cells to 15,700; the thoracic roentgenogram showed widening of the superior mediastinum (Fig. 1); the electrocardiogram demonstrated an auricular fibrillation. Esophagoscopy performed under local topical anesthesia on June 12 showed a phlegmonous cellulitis of the hypopharynx and a large pharyngoesophageal diverticulum in which edema, ulcerations, and a thick green, moldy exudate were present. The esophageal opening was completely obscured by an edematous inflammatory reaction but was found by the passing of a small filiform

Read at Wisconsin Surgical Society Meeting, October, 1954.
Received for publication Jan. 12, 1955.

Jackson bougie. A small (4 by 30) scope was passed over the bougie into the upper esophagus and an ulcerative esophagitis with tremendous edema was seen in the upper 3 inches. A thread was passed into the lower esophagus through the scope for later dilatations.

The patient was prepared by parenteral antibiotics, intravenous fluids, irrigations of the diverticulum, and dilatations of the esophagus over the previously passed string. On June 19, the superior mediastinum was explored through a collar incision in the neck exposing the visceral compartment on the right side of the neck. A large, well-walled-off abscess filled with pus was found in the superior mediastinum, caused by a perforation of the diverticulum by a triangular piece of beef bone. The abscess cavity was evacuated and cleansed of foreign

Fig. 1.—Posteroanterior roentgenogram of chest showing widening of the superior mediastinum.

matter. The diverticulum was lifted with ease from the periesophageal structures, the opening in the hypopharynx was closed with a single row of interrupted 0000 cotton sutures, and this area was reinforced by closing the inferior constrictor and cricopharyngeus muscles over the same region. Drains from the abscess area were brought out the right lateral corner of the wound and the wound was closed.

The patient showed an immediate favorable response postoperatively, with the temperature and pulse dropping to normal within twenty-four hours. On the fourth postoperative day a clear liquid diet was started after a Lipiodol swallow had shown no roentgen evidence of leak from the diverticular closure site. On the seventh postoperative night the patient accidentally broke her esophageal thread guide and she refused the house staff's request to start another thread at that time. She continued taking a liquid diet, however, until the tenth postoperative day when she stated she was unable to swallow, and this condition persisted.

On July 1, esophagoscopy was performed and the closure wound of the diverticulum was visualized and appeared well healed, but the lumen of the esophagus below this level showed a gradual narrowing, and at a level of the cricopharyngeus no opening could be found. Two days later a gastrostomy was performed and attempts to insert tubes, bougies, and a thread retrograde to the pharynx were unsuccessful. The patient's recovery from these procedures was uneventful and she showed a progressive improvement with cervical wound healing and progressive weight gain under gastrostomy feedings. She continued unable to swallow, however, and regurgitated even water with attempted ingestion.

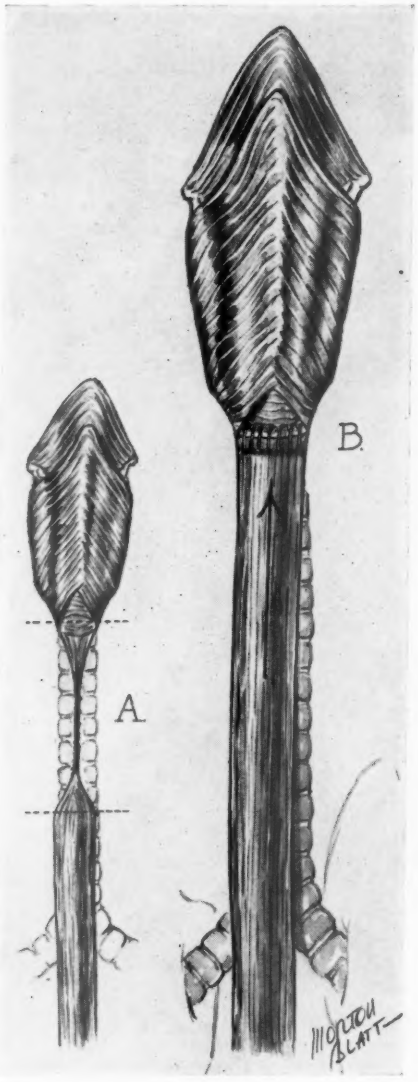


Fig. 2.—Artist's diagrammatic representation of pathology found and surgery performed. Upper esophagus obliterated by inflammatory reaction. Reanastomosis of lower esophagus to pharynx.

The neck was re-explored on July 31, utilizing a secondary transverse incision excising the previous scar. The upper esophagus and hypopharynx were exposed on both sides by elevation of the thyroid lobes and retraction of the trachea and carotid sheaths. The upper esophagus was found to be replaced for a distance of about 2 inches below the cricopharyngeus

area by a thick fibrous cord, and no lumen could be identified within this cordlike structure. The area of the previous diverticulum closure above this lesion was well healed and there was no significant residual of the former abscess cavity.

It was found that the thoracic esophagus could be freed from the surrounding structures by blunt dissection down to the aortic arch level. By dissecting the pharyngeal mucosa from the posterior larynx and the posterior pharyngeal wall, it could be pulled down to where it could be anastomosed to the normal distal esophagus. Therefore, the obliterated esophageal segment was excised and the intrathoracic esophagus anastomosed to the pharyngeal mucous membrane under moderate tension, using a single layer of interrupted 0000 cotton sutures (Fig. 2). A No. 36 Fr. endotracheal tube was passed through the anastomosis to be utilized as a splint and was sutured to the inside of the left cheek. A tracheotomy was performed. The wound was closed with drainage of the visceral compartment after closing the inferior constrictor and cricopharyngeus muscles around the anastomosis. A splint was applied to keep the head and neck flexed on the chest and this was replaced in four days by a plaster cast to the head, neck, and chest for the same purpose.

Fig. 3.—Esophagogram made ten days following anastomosis of lower esophagus to pharynx.

The patient's convalescence was not unusual and she was fed through the gastrostomy tube. The esophageal splint was removed, cleansed, and replaced on the fifth postoperative day and anchored at this time to the outside of the left cheek by adhesive tape and a large silk suture sewn through the tube. The esophageal splint was removed on the tenth postoperative day, the plaster cast discarded, and a liquid diet started by mouth after a thin barium swallow had shown no evidence of an anastomotic leak (Fig. 3).

Soft foods were gradually introduced into the patient's diet, but this was done slowly because she had difficulty in coordinating her swallowing movements. A No. 46 Fr. bougie

was passed through the anastomosis weekly for three months to check the patency of the lumen, and she gradually returned to a general diet and returned to home two months after the last operation. The gastrostomy tube was removed three months postoperatively and the sinus tract healed promptly. The tracheotomy tube was plugged and removed a month later, and a small tracheal sinus tract was excised and the neck wound closed.

The patient's course has continued to be satisfactory; she gained 27 pounds during the first four months postoperatively and she has maintained that weight. Follow-up roentgen studies have shown moderate spasm and narrowing in the area of the anastomosis but this has not been persistent (Fig. 4). At monthly examinations for six months, a No. 50 Hurst dilator was passed into the esophagus with comparative ease. The patient had no particular complaints when examined one year after her resection and anastomosis, although she stated that she had to eat slowly, chew her food well, and concentrate on the swallowing act because of a fear of choking, a conscious effort being needed to initiate the act.

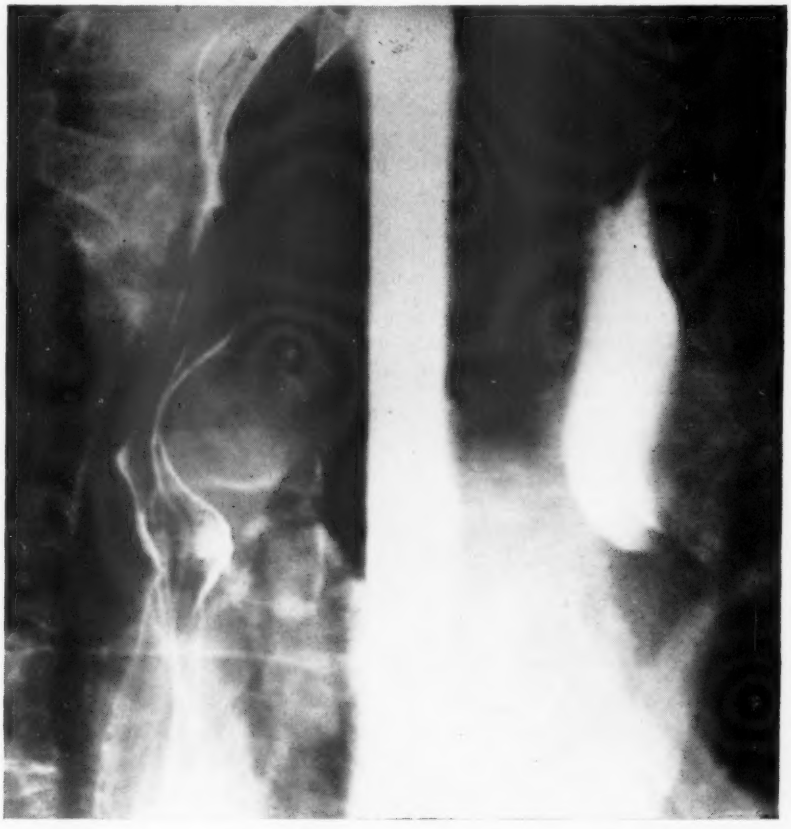


Fig. 4.—Postoperative oblique roentgenograms of the esophagus showing moderate spasm in the new upper esophageal segment.

DISCUSSION

Although limited resections of the esophagus have been described, we have not found a patient reported in whom the upper esophagus was resected and the lower segment reanastomosed to the pharynx. That this can be accomplished by observing the surgical principles already established has been demonstrated by the case herein reported.

In addition, numerous complications have been recorded which have arisen in conjunction with esophageal diverticula. The occurrence, however, of an obliterative esophagitis as a sequelae to the perforation of a diverticulum has apparently been most unusual.

SUMMARY AND CONCLUSION

1. A patient has been reported in whom a pharyngoesophageal diverticulum was perforated by an ingested foreign body, and she developed, as a complication, an obliterative esophagitis of the upper esophagus.

2. For treatment, the patient required a resection of the upper esophagus, and the distal segment was successfully reanastomosed to the pharynx.

REFERENCES

1. Adams, W. E.: Discussion of reference 18.
2. Barnes, W. A., and McElwee, R. S.: Surgical Treatment of Non-Neoplastic Lesions at the Esophago-Gastric Junction, Ann. Surg. **137**: 523-529, 1953.
3. Baronofsky, I. D., and Hilger, J.: Fascia Lata Transplants for Resected Cervical Esophagus, SURGERY **30**: 355-360, 1951.
4. Berman, E. F.: The Experimental Replacement of Portions of the Esophagus by a Plastic Tube, Ann. Surg. **135**: 337-343, 1952.
5. Berman, E. F.: A Plastic Prosthesis for Resected Esophagus, Arch. Surg. **65**: 916-919, 1952.
6. Burnett, W. E.: Discussion of reference 24.
7. Gross, R. E.: Treatment of Short Stricture of the Esophagus by Partial Esophagectomy and End-to-End Esophageal Reconstruction, SURGERY **23**: 735-744, 1948.
8. Kay, E. B., and Brown, K. L.: Partial Esophagectomy With End-to-End Anastomosis, SURGERY **29**: 658-662, 1951.
9. Kleinsasser, L. S., Cramer, I., and Warshow, H.: Anastomosis of the Cervical Esophagus. Experimental Evaluation of Peritoneal Grafts, SURGERY **28**: 438-442, 1950.
10. Klopp, C. T., Alford, C., and Pierpont, H.: The Use of Polyethylene Film and Split-Thickness Skin Graft in Reconstruction of Cervical Esophageal and Pharyngeal Defects, SURGERY **29**: 231-239, 1951.
11. Maier, H.: Discussion of reference 24.
12. Paine, J. R.: Discussion of reference 18.
13. Parker, E. F., and Brockington, W. S.: Esophageal Resection With End-to-End Anastomosis: Experimental and Clinical Observations, Ann. Surg. **129**: 588-605, 1949.
14. Parker, E. F.: Discussion of reference 18.
15. Postlethwait, R. W., Deaton, W. R., Bradshaw, H. H., and Williams, R. W.: Esophageal Anastomosis: Types and Methods of Suture, SURGERY **28**: 537-543, 1950.
16. Saint, J. H., and Mann, F. C.: Experimental Surgery of the Esophagus, Arch. Surg. **18**: 2,324-2,338, 1929.
17. Shapiro, A. L., and Robillard, G. L.: The Esophageal Arteries, Ann. Surg. **131**: 171-185, 1950.
18. Shek, J. L., Prietto, C. A., Tuttle, W. M., and O'Brien, E. J.: An Experimental Study of the Blood Supply of the Esophagus and Its Relation to Esophageal Resection and Anastomosis, J. Thoracic Surg. **19**: 523-533, 1950.
19. Strieder, J. W.: Surgery of the Esophagus, New England J. Med. **342**: 445-453, 1950.
20. Sweet, R. H., Robbins, L. L., Gephart, T., and Wilkins, E. W.: The Surgical Treatment of Peptic Ulceration and Stricture of the Lower Esophagus, Ann. Surg. **139**: 258-268, 1954.
21. Swenson, O., and Clatworthy, H. W.: Partial Esophagectomy With End-to-End Anastomosis in the Posterior Mediastinum; An Experimental Study, SURGERY **25**: 839-848, 1949.
22. Swenson, O., Merrill, K., Peirce, E. C., and Rheinlander, H. F.: Blood and Nerve Supply to the Esophagus, J. Thoracic Surg. **19**: 462-476, 1950.
23. Swenson, O.: Discussion of reference 9.
24. Tuttle, W. M., and Day, J. C.: The Treatment of Short Esophageal Strictures by Resection and End-to-End Anastomosis, J. Thoracic Surg. **19**: 534-541, 1950.

TUBERCULOUS EROSION OF THE RIGHT COMMON ILIAC ARTERY WITH RUPTURE INTO THE DUODENUM

A Case Report and Further Tabular Review of Rupture of the Aorta Into the Gastrointestinal Tract

William S. Reed, M.D., Ann Arbor, Mich.

(From the Departments of Surgery and Pathology, University of Michigan Medical School)

AN EXAMPLE of the rupture of a tuberculous false aneurysm of the upper portion of the right common iliac artery into the duodenum provided the impetus for further consideration of the clinicopathologic syndrome produced by the rupture of an aortic lesion into the gastrointestinal tract. While the lesion in the patient to be described was not in the aorta anatomically, it was situated so close to the bifurcation that the clinical effect was entirely similar to rupture of the aorta itself. Therefore, it seemed appropriate to continue the tabular review of such cases, which was initiated by Rottino[1] and enlarged by Hunt and Weller[2] and by Scott, Maxwell, and Grimes.[3]

CASE REPORT

A widow, aged 70, was admitted to the University Hospital because of bleeding from the gastrointestinal tract. Five months before admission, she had had oliguria for two days culminating in gross hematuria with passage of blood clots. No further hematuria occurred, but two weeks later she complained of severe epigastric pain accompanied by faintness, nausea, and vomiting of coffee-ground material. She also developed melena and passed red blood in the stool. Within three days she had recovered and again felt well. Three weeks prior to admission to the University Hospital she again had severe epigastric pain, became faint, and passed approximately 3 pints of bright red blood. She was hospitalized in a local hospital and blood transfusions were given. She again gained strength and upon admission to this hospital felt well.

She had had a chronic cough for twenty years, with many roentgenograms and sputum studies for tuberculosis which were not diagnostic.

On physical examination the patient was found to be well nourished and in no acute distress. Blood pressure was 138/78 mm. Hg and pulse rate 76 per minute. The abdomen was soft and a mass was not palpated. The hemoglobin was 13.5 Gm. per 100 c.c. of blood. The Kahn serologic reaction was negative. A 4 plus guaiac test was obtained on the stool specimen. Tuberculin skin test was equivocal in 1:10,000 dilution. Three serial sputum examinations were negative for acid-fast bacilli. The prothrombin concentration was 100 per cent. Aspiration of marrow from the right iliac crest revealed no neoplastic cells. Complete hematologic studies gave normal findings. Gastroscopic examination revealed no lesion, and upper gastrointestinal examination with barium showed no abnormality. A cholecystogram indicated multiple small gallstones and, by barium enema, colonic diverticula were demonstrated. Retrograde pyelograms and cystoscopic examination showed no abnormality.

A laparotomy was performed to determine, if possible, the cause of the gastrointestinal bleeding. Careful examination of the alimentary tract showed no abnormality other than

Received for publication Jan. 24, 1955.

cholelithiasis. The duodenum and aorta were examined and were believed to be normal. The stomach was smooth and pliable, and it was decided that gastric resection was not indicated. The gall bladder was removed and the common bile duct explored. Specimens for biopsy were secured from the liver and from the right iliac crest. The patient did well postoperatively until the ninth day, when she complained of severe epigastric pain and fainted after eating breakfast. She then passed tarry and bloody stools. The following morning she had massive hematemeses and, although given 25 pints of blood by vein, she expired.

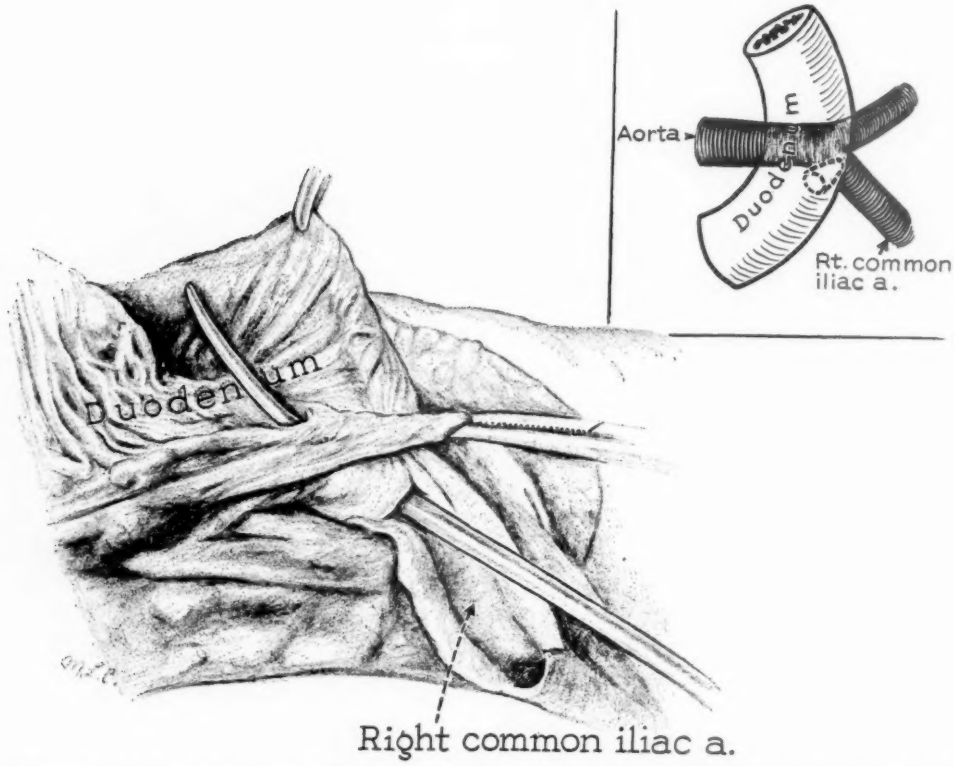


Fig. 1.—A drawing, prepared from a photograph, shows a probe passed from the first portion of the right common iliac artery through the eroded false aneurysm into the duodenum. The small sketch shows the position of these structures before they were disturbed.

Necropsy Findings.—At necropsy a small perforation, 4 mm. in diameter, was found at the junction of the middle and distal thirds of the duodenum (Fig. 1). The duodenum was adherent to the right common iliac artery in this area, and at a point 2.2 cm. below the bifurcation of the aorta on the medial aspect of the common iliac artery, there was a small ostium, 5 mm. in diameter, within an atheromatous plaque. The sinus formed between the two openings was 10 mm. in length and 4 mm. in diameter. The stomach and small and large intestines were distended with blood, and blood had been aspirated into the lungs. The lungs revealed bilateral pulmonary fibrosis.

On microscopic examination the fistula between the right common iliac artery and ascending portion of the duodenum was found to be inflammatory and granulomatous. Caseating granulomatous tissue extended into the duodenum. There was advanced anthracosis of the lungs and bronchial lymph nodes with slight anthracosis of the spleen and liver. Multiple miliary granulomatous lesions were present in the lungs, lymph nodes, spleen, and liver. Tuberculosis, sarcoidosis, and systemic silicosis were considered in differential diagnosis. The granulation tissue about the fistula showed the best evidence for

tuberculosis of all sections examined. Staining of sections for tubercle bacilli was done with negative results. This did not disprove the etiological diagnosis of tuberculosis, but left it resting largely on the histologic findings.

Initially, this patient complained of oliguria lasting for two days and followed by hematuria. No evidence of disease of the urinary tract was found clinically or at necropsy. The right ureter was immediately adjacent to the area of perforation, and it is conceivable that pressure upon the ureter could have caused the symptoms listed. Perforation of the common iliac artery into the ureter due to mycotic aneurysm has been described.[4]

Uhle[5] described the urologic problems in abdominal aneurysm in a comprehensive review. Aneurysm may simulate perirenal abscess, renal tumor, and perirenal hemorrhage from trauma and may cause hematuria, hydronephrosis, uremia, and anuria. These result from (1) hemorrhage into the perirenal areas following rupture of the sac, (2) displacement of the kidney and ureter by the hemorrhage or the unruptured sac, and (3) a combination of vertebral erosion and nerve root pressure in the lower thoracic and lumbar areas.

Involvement of the aorta by a tuberculous process is relatively rare. It may result in three ways, as described by Dafoe[6]: (a) direct hematogenous implantation on the internal surface where slight injury or other change has occurred, (b) transportation of the organisms to the adventitia or media by the vasa vasorum, and (c) direct extension from tuberculous lymph gland or abscess. In most instances of tuberculous aneurysm the continuity of the vessel wall cannot be established in the aneurysmal sac. Such aneurysms are therefore false aneurysms. Gellerstedt and Säfwenberg[7] reviewed the problem and reported 20 cases from the literature, to which they added 1 case of their own. Of these, 14 were false aneurysms, 2 were of mixed type, 2 were dissecting, and 3 were not described as to type. The case reported by Frosch and Horowitz[8] was also one of tuberculous false aneurysm. Similarly, cases described by Scott, Maxwell, and Grimes,[3] Owens and Bass,[9] Cade, Dechaume, and Grandmaison,[10] Bagnuolo and Bennett,[11] and Herndon, Galt, and Austin[12] were of this type.

In almost all instances tuberculous aneurysm results from erosion of the vessel wall by necrotizing inflammation from adjacent caseous lymph nodes. When the vessel ruptures, either a false-aneurysmal, blood-filled space is formed or massive bleeding occurs into whatever serous cavity or viscus may be contiguous or adherent, including the esophagus, stomach, and intestinal tract. Tuberculous aneurysm occurs with about equal frequency in the upper aorta (ascending portion and arch) and in the abdominal aorta. In the abdominal aorta the portion between the diaphragm and the celiac axis was involved in 9 of 12 reported cases.[3, 7-10] In this area, aneurysms may rupture into the stomach. In the lower abdominal aorta rupture may occur into the duodenum or jejunum, the former being the more common site.

Rottino[1] presented a series of 31 cases of aneurysm of the abdominal aorta with rupture into the gastrointestinal tract, to which he added 1 of his own. Hunt and Weller[2] increased the series to 41, using the tabular presentation of Rottino. Scott, Maxwell, and Grimes[3] added 4 cases. In connection with the

TABLE I. AORTIC ANEURYSM RUPTURING INTO THE GASTROINTESTINAL TRACT

NO.	DATE	AUTHOR	AGE	SEX	PERTINENT HISTORY LEADING TO ADMISSION	CLINICAL OBSERVATIONS	COURSE	PATHOLOGIC CHANGES
46*	1893	Perry and Shaw[13]	37	M	Large abdominal mass present for 14 months	Palpable abdominal mass	Sudden death during hospitalization	In distal duodenum a small orifice opened into the sac of a median aneurysm of the abdominal aorta
47	1927	Cade, Dechaume, and Grandmaison[10]	63	M	Painful dyspepsia, hematemesis, and melena for 1 week; pleurisy 20 years prior to admission; continuous violent abdominal pain 8 days prior to admission	Negative physical examination; marked anemia	Continued to have abundant hemorrhagic stools; death 5 days after admission	Bowel full of blood; adhesions between 3rd portion of duodenum and aorta; communication between aorta and duodenum through an area of tuberculous caseation necrosis; aortic atheromas
48	1943	Hausmann[14]	79	F	Pain in vulva and rectum for 7 years; rectal hemorrhage 14 days prior to admission	Palpable tumor in the upper abdomen to the left of the midline	Death 6 days after admission following the onset of hematemesis	Saccular aneurysm of the lower abdominal aorta behind the pancreas, 7 cm. in diameter, communicating with the jejunum through an aperture 5 mm. in diameter; aortic atheromatosis
49	1944	Frosch and Horowitz[8]	51	M	Tuberculous epididymitis 6 years prior to admission; developed hematemesis immediately prior to final admission to hospital	Draining sinuses over the sternum; stool positive for blood	Sudden death on day following discharge from hospital; massive hematemesis	Communication ¼ inch in diameter from the abdominal aorta to the 3rd portion of the duodenum through a caseous tuberculous lymph node
50	1945	Irwin and Frankel[15]	60	M	Pain in shoulders, back, and legs; difficulty in swallowing solids; shooting pains down both legs	Loud blowing systolic-diastolic murmur; aortic aneurysm noted on x-ray	Death 16 days after admission; developed melena; complained of sudden precordial pain and developed shock	Saccular aneurysm originating in the upper 2 inches of the abdominal aorta, perforating the posterior wall of the gastric fundus; syphilitic aortitis
51	1948	Baer and Loewenberg[16]	?	F	Back pain for 2 months; hematemesis 2 weeks prior to admission; 30 lb. weight loss	Epigastric mass	Sudden death day after admission	Abdominal aortic aneurysm with rupture into the first portion of the duodenum; gastric carcinoma

*Numbered in sequence with cases reported by Rottino,[1] Hunt and Weller,[2] and Scott, Maxwell, and Grimes.[3]

52	1948	Klein and Bardos[17]	61	M	Diagnosis of heart disease 1 year before death; long history of asthma; 3 months prior to admission developed pneumonia; epigastric pain with radiation to sacrum	Not stated	Hematemesis day before death; found dead in bed, having again vomited blood	Old tuberculous scarring of right pulmonary apex; adhesions of the descending portion of the duodenum to the aorta; 1.5 cm. above the bifurcation an opening 30 mm. by 20 mm. communicated with the duodenum 2 cm. below the ampulla; aortic atheromatosis
53	1949	Pomerantz[18]	53	F	3 weeks' history of recurrent severe left abdominal pain	Mid-epigastric mass	Death 2 days after admission with recurrence of pain, development of hematemesis and shock	Saccular aneurysm of the aorta with rupture into the jejunum 3 inches below the ligament of Treitz
54	1949	Barrett[19]	58	F	Anorexia and abdominal discomfort for 5 months; hematemesis 12 hours prior to admission	Abdominal tumor in the region of the umbilicus	Death 1 day following admission shortly after onset of 2nd episode of hematemesis	Saccular aneurysm arising from the abdominal aorta with perforation into the 3rd portion of the duodenum; aortic atheromas
55	1950	Balakian, Ryan, and Perkel[20]	59	M	1½ hours prior to admission patient developed melena and inability to move left leg; right leg amputated 10 years before for osteogenic sarcoma	Blood pressure 115/65; nontender pulsating abdominal mass slightly below the umbilicus	Recurrence of rectal bleeding 24 hours after admission, death	Aneurysm of the abdominal aorta just below the superior mesenteric artery, with erosion of adjacent lumbar vertebrae; perforation into the 3rd portion of the duodenum
56	1951	Hirst and Affeldt[21]	66	F	High blood pressure for 2 years; unexplained weakness for 10 days prior to admission; pulsating abdominal mass for 30 years	Blood pressure 200/105; 12.5 cm. smooth, firm, pulsating, expansile mass in mid-abdomen	28 hours after admission, vomited blood and passed bloody stools; shock developed and death occurred 20 hours later	Saccular aneurysm of abdominal aorta above the bifurcation perforating into 3rd portion of the duodenum; aortic atheromatosis
57	1951	Hirst and Affeldt[21]	67	M	Hematemesis 2 days prior to admission, with recurrence of bleeding 4 hours prior to admission	Nontender pulsating mass to the right of the umbilicus	Went into shock 11 hours after admission and died 25 minutes later	Arteriosclerotic aneurysm of the abdominal aorta, 8 by 5 cm., perforating into the third part of the duodenum

TABLE I—CONT'D

NO.	DATE	AUTHOR	AGE	SEX	PERTINENT HISTORY LEADING TO ADMISSION	CLINICAL OBSERVATIONS	COURSE	PATHOLOGIC CHANGES
58	1951	Hirst and Affeldt[21]	65	M	Abdominal mass for 19 months prior to admission; immediately before admission developed epigastric pain referred to the left lumbar area	Hard, slightly tender, 10 cm. epigastric mass	Left hospital in 14 days unimproved; readmitted a few days later in shock and expired in 2 hours	Arteriosclerotic aneurysm originating just below the renal vessels, 8 cm. in diameter, with perforation into the 3rd portion of the duodenum; severe atheromatosis of the aorta
59	1951	Hirst and Affeldt[21]	80	F	Epigastric pain, tarry stools, and hematemesis for 24 hours	Shock; 8 by 8 cm. firm, nonmovable, pulsating, epigastric mass	Died 8 hours after admission	5 cm. saccular aortic aneurysm extending downward toward the bifurcation; perforation into 3rd portion of duodenum
60	1951	Hirst and Affeldt[21]	76	M	Burning pain in left lower quadrant, referred to the back for 3 weeks; syncope 6 hours prior to admission followed by melena and hematemesis	Tenderness and increased resistance in the left lower quadrant	Expired 46 hours after admission	7 cm. below the superior mesenteric artery the aorta had perforated anteriorly through an atheromatous area in continuity with a 3 mm. perforation in the 3rd portion of the duodenum
61	1951	Hirst and Affeldt[21]	73	F	Past history of hypertension and positive Wassermann test; gnawing upper right quadrant pain, anorexia, nausea, vomiting, and weakness of 11 days' duration	Large, irregular, slightly tender, pulsating mass in the left upper quadrant	Discharged 2 days after admission; returned 2 months later with hematemesis and melena; died in a few days	Aortic aneurysm 7 cm. in diameter arising 2 cm. distal to the origin of the renal arteries, and communicating with a 7 mm. perforation in the 3rd part of the duodenum; syphilitic aortitis
62	1951	Hirst and Affeldt[21]	75	M	Diabetes mellitus for 10 to 15 years; urinary frequency and drowsiness for 2 days; colicky abdominal pain and vomiting for 12 hours	Rounded, nontender, epigastric mass extending into the right half of the abdomen	5 days after admission vomited a large quantity of blood and expired	10 cm. aortic aneurysm at the level of the 4th and 5th lumbar vertebrae; the duodenum was perforated in four places into the aneurysmal sac; aortic atherosclerosis
63	1951	Hirst and Affeldt[21]	78	M	Abdominal pain and melena for 1 month	Pulsating, nontender, epigastric mass	Melena continued; on 6th day developed massive hematemesis and died	5 by 7 cm. arteriosclerotic aneurysm communicating with a 1 cm. perforation in the duodenum 10 cm. below the pylorus

64	1951	Antzis, Dunn, and Schilero[22]	78	F	Dyspnea, weakness, precordial distress	Expansile, right upper quadrant, sausage-shaped mass (noted 4 months after admission)	2 months after discovery of mass, patient was found on bathroom floor semiconscious; she vomited blood and passed bloody stools; expired 31 hours after onset of hematemesis	Below the renal arteries an aneurysm the size of an egg lay beneath the proximal portion of the superior mesenteric artery; perforation into the 3rd portion of the duodenum through a 1.5 mm. opening; atherosclerosis of aorta
65	1952	Sandrock, Emerson, and Emerson[23]	46	F	Far-advanced pulmonary tuberculosis with bilateral cavitation diagnosed 14 months before death; 5 months before death developed abdominal distention, pain, and fever; therapeutic pneumoperitoneum	Abdomen distended; râles over lungs; clubbing of digits	1 month prior to death developed constant lower abdominal pain with circle radiation to back; passed "currant jelly" stools and vomited blood, starting 3 hours before death	Tuberculous false aneurysm of abdominal aorta 2 cm. above the bifurcation, communicating with the first part of the jejunum by two fistulas, the larger being 1.5 cm. in diameter; tubercles in spleen, liver, and kidney
66	1952	Hirst and Abbott[24]	72	M	Nausea and abdominal distention for 1 week; developed hematemesis and melena in morning; coffee-ground emesis at night; collapsed in bathroom following morning when admitted	Shock; diffuse epigastric tenderness; no mass	Hematemesis and melena continued; died on 3rd hospital day	Saccular aneurysm 3 cm. in diameter and 6 cm. in length just above bifurcation; perforation 1 cm. in diameter into 3rd portion of duodenum 2 cm. distal to ampulla of Vater
67	1954	Reed	70	F	Oliguria; hematuria; severe epigastric pain; faintness; coffee-ground emesis; melena; lost 3 pints of bright red blood by rectum; chronic cough for 20 years, tuberculosis suspected, but unproved	No mass palpable; gastric studies negative	Laparotomy done in search for cause of bleeding; hematemesis and melena recurred on 9th postoperative day	Tuberculous false aneurysm of right common iliac artery immediately below aortic bifurcation; perforation into distal third of duodenum

present report the literature has been reviewed and 21 cases have been added. With the 1 described in this paper, the total is now 67. All cases have been numbered serially with the original series (Table I).

In the total series the sex distribution is predominantly male, in a proportion of approximately 3 to 1. In age, the patients varied from 20 to 81 years. The duodenum was the site of rupture in 52 instances, with the third portion being involved in 45 cases. The first and second portions of the duodenum were each involved once. In 5 cases the portion of the duodenum involved was not indicated. Rupture into the jejunum occurred 6 times and into the stomach, 7 times. In 7 cases a tuberculous aneurysm was present. The ages of patients in this subgroup varied from 32 to 70 years, all but 2 being 49 or older.

The clinical syndrome which results when an aneurysm of the aorta or of one of its major branches ruptures into the gastrointestinal tract has been described repeatedly and needs no detailed elaboration. The combination of massive hematemesis, melena, and hemorrhagic shock should always suggest this possibility, and prompt diagnostic studies may make operative intervention possible. In about 50 per cent of all cases, the first massive hemorrhage does not culminate in death. During the interval between the first and subsequent hemorrhages an opportunity for diagnostic studies may be afforded. An expansile, pulsating abdominal mass may be noted and a bruit and thrill may be discovered. The combination of severe epigastric or lumbar pain, urologic symptoms, and complaints of discomfort of vascular or neurogenic type in the lower extremities is common enough to stimulate the consideration of aneurysm. If radiologic studies do not demonstrate an abnormality of the gastrointestinal tract and if an abdominal mass or vertebral erosion is not seen, aortography may be useful.

If surgical exploration is done, it is important to recall that when bleeding into the bowel occurs due to aneurysmal rupture, (1) the most frequent site of such bleeding is the third portion of the duodenum; (2) more rarely, the stomach and upper jejunum may be the site; (3) when rupture occurs into the duodenum or jejunum, the aneurysm will be located in the abdominal aorta below the superior mesenteric artery, and (4) one of the branches of the aorta may be the site of the aneurysm. If an aortic aneurysm is not demonstrated and external examination of stomach and intestines reveals no abnormality, the stomach should be opened and a search made for ulcer, neoplasm, and varices. At this time, search may be extended to include cirsoid and congenital aneurysm of the stomach.

SUMMARY

Twenty-two cases of rupture of an aneurysm of the abdominal aorta into the gastrointestinal tract have been added to the series of Rottino,[1] Hunt and Weller,[2] and Scott, Maxwell, and Grimes,[3] bringing to 67 the number of cases known to have been reported.

A case is presented which is similar to the others except that perforation into the duodenum occurred from the right common iliac artery just below

the bifurcation of the aorta. This perforation was caused by erosion through a tuberculous lymph node which had become adherent to both structures, and the resulting fistula was surrounded by tuberculous granulation tissue.

REFERENCES

1. Rottino, A.: Aneurysm of the Abdominal Aorta, With Rupture Into the Duodenum. Case Report and Review of the Literature, Am. Heart J. **25**: 826, 1943.
2. Hunt, H. H., and Weller, C. V.: The Syndrome of Abdominal Aortic Aneurysm Rupturing Into the Gastrointestinal Tract. Summary of the Literature and Case Report, Am. Heart J. **32**: 571, 1946.
3. Scott, J. W., Maxwell, E. S., and Grimes, A. E.: Tuberculous False Aneurysm of Abdominal Aorta With Rupture Into the Stomach. A Case Report With Review of the Literature, Am. Heart J. **37**: 820, 1949.
4. Taylor, W. N., and Reinhart, H. L.: Mycotic Aneurysm of Common Iliac Artery With Rupture Into Right Ureter, J. Urol. **42**: 21, 1939.
5. Uhle, C. A. W.: The Significance of Aneurysm of the Abdominal Aorta Masquerading as Primary Urologic Disease, J. Urol. **45**: 13, 1941.
6. Dafoe, W. A.: Ruptured Aneurysms of the Abdominal Aorta Due to Tuberculosis. A Report of Two Cases, Edinburgh M. J. **32**: 291, 1925.
7. Gellerstedt, N., and Säfwenberg, O.: Zur Kenntnis der Aortatuberkulose und des tuberkulösen Arrosionsaneurysmas der Aorta, Upsala läkaref. förh. **38**: 165, 1933.
8. Frosch, H. L., and Horowitz, W.: Rupture of Abdominal Aorta Into Duodenum (Through a Sinus Tract Created by a Tuberculous Mesenteric Lymphadenitis), Ann. Int. Med. **21**: 481, 1944.
9. Owens, J. N., Jr., and Bass, A. D.: Tuberculous Aneurysm of the Abdominal Aorta. Report of a Case, Arch. Int. Med. **74**: 413, 1944.
10. Cade, Dechaume, J., and Grandmaison: Periaortite caséeuse ayant entraîné l'ouverture de l'aorte dans la troisième portion du duodénum, Lyon méd. **139**: 103, 1927.
11. Bagnuolo, W. G., and Bennett, H. D.: Nontraumatic Aortic Perforations Into Gastrointestinal Tract. Review of the Literature and Report of an Unusual Case, Am. Heart J. **40**: 784, 1950.
12. Herndon, J. H., Galt, J., and Austin, D. J.: Ruptured Tuberculous False Aneurysm of the Abdominal Aorta. Report of a Case With Resection of the Aneurysm and Survival for Six Days, Texas J. Med. **48**: 336, 1952.
13. Perry, E. C., and Shaw, L. E.: On Diseases of the Duodenum, Guy's Hosp. Rep. **50**: 307, 1893.
14. Hausmann, W.: Arteriosclerotic Aneurysm of Abdominal Aorta With Perforation Into Upper Jejunum, Brit. M. J. **2**: 362, 1943.
15. Irwin, D. H., and Frankel, E.: An Unusual Case of Ruptured Aortic Aneurysm, Brit M. J. **2**: 425, 1945.
16. Baer, S., and Loewenberg, S. A.: Aortic Aneurysms Simulating Organic Disease of the Gastro-Intestinal Tract, Gastroenterology **10**: 617, 1948.
17. Klein, F., and Bardos, A.: Ueber eine Ruptur der Bauchaorta in den absteigenden Schenkel des Zwölffingerdarms, Schweiz. Ztschr. Path. u. Bakt. **11**: 387, 1948.
18. Pomerantz, R. B.: Abdominal Aneurysm With Aorto-Jejunal Rupture. Fourth Reported Case, Am. Heart J. **37**: 142, 1949.
19. Barrett, G. M.: Haematemesis Following Intraduodenal Rupture of Aneurysm, Brit. M. J. **2**: 319, 1949.
20. Balakian, G., Ryan, C., and Perkel, L. L.: Gastrointestinal Hemorrhage Due to Ruptured Abdominal Aortic Aneurysm, Am. J. Digest. Dis. **17**: 408, 1950.
21. Hirst, A. E., Jr., and Affeldt, J.: Abdominal Aortic Aneurysm With Rupture Into the Duodenum. A Report of Eight Cases, Gastroenterology **17**: 504, 1951.
22. Antzis, E., Dunn, J., and Schilero, A. J.: Rupture of Abdominal Aneurysm Into the Gastrointestinal Tract, Am. J. Med. **11**: 531, 1951.
23. Sandrock, R. S., Emerson, G. L., and Emerson, M. S.: A Rare Complication of Tuberculous Mesenteric Lymphadenitis. A Case of Fatal Hemorrhage From Abdominal Aorta Into Jejunum Through a Tuberculous Lymph Node, Am. Rev. Tuberc. **65**: 210, 1952.
24. Hirst, A. E., and Abbott, A. W.: Aneurysm of the Abdominal Aorta With Rupture Into the Duodenum, California Med. **77**: 197, 1952.
25. Reed, William S.: The Causes of Massive Gastrointestinal Hemorrhage as Observed in Necropsy Material, Univ. Michigan M. Bull. 20: 62-65 (March) 1954.

ACUTE CHYLOUS ASCITES IN INFANCY

Report of Case in Which the Cause Was Mesenteric Lymphadenitis

Sam J. Kuykendall, M.D.,* and Alexander Dederer, M.D.,**
Fort Worth, Tex.

(From Carswell Air Force Hospital)

THE rarity of acute chylous ascites in infancy and childhood is evidenced by the paucity of reports in the literature. Gribetz and Kanof (1951)[2] reviewed the world literature and found only 25 cases of chylous effusion into the peritoneal cavity among children 15 years of age or younger. Of these patients, only five presented the signs and symptoms of an acute abdomen. Hoffman (1954)[3] reported on 24 children and adults in whom free chyle was found in the peritoneal cavity at either laparotomy for an acute abdominal emergency or at necropsy. His series included one instance of acute chylous ascites from this group, in addition to three other instances in children.

We have been able to find no other case in the literature since Hoffman's report. Therefore, the present report concerns the ninth recorded case of acute chylous ascites with free chyle in the peritoneal cavity in infancy or childhood, and the second recorded case in which acute chylous ascites is presumed to be secondary to acute mesenteric lymphadenitis. Davis[1] reported a similar case in childhood with free chylous ascites, and two others in which a chyle cyst secondary to enlarged mesenteric glands was found. All his patients were 4 years of age or older at the time of diagnosis.

Acute chylous ascites must be distinguished from the relatively more common chronic chylous ascites which may or may not be associated with effusion into the pleural or pericardial sacs. The chronic form may be due to a variety of conditions, such as traumatic rupture of the thoracic duct or lacteals, obstruction of the great veins in the neck, or obstruction of the thoracic duct, with resultant extravasation of chyle. The latter may be produced by tumors, enlarged lymph nodes, or parasitic obstruction, as in filariasis.

CASE REPORT

A 10-day-old Negro male infant was admitted to an Air Force hospital on Dec. 10, 1953, with a 2-day history of progressive right scrotal enlargement. The parents had noted no change in the size of the abdomen. The infant apparently was nursing, voiding, and defecating normally. He was the product of a first pregnancy which was full-term and uncomplicated. Delivery was uneventful and atraumatic. The mother gave no history of injury. Both parents were healthy.

The infant was small, with moderate abdominal enlargement symmetrically, and with signs of a distended bowel and free fluid in the peritoneal cavity. The right side of

Received for publication March 7, 1955.

*Fellow in Surgery, Mayo Foundation, Rochester, Minn.
**Present address: Eugene, Oregon.

the scrotum was 5 cm. in diameter and tense; the overlying skin was thin and shiny. There was no fluid wave, and the scrotal contents could not be reduced into the peritoneal cavity. Results of erythrocyte and leukocyte counts, of urinalysis, and the value for hemoglobin were within normal limits. A roentgenogram of the abdomen was not made.

At operation the right inguinal canal was explored through an incision in the groin. Structures coursing through the canal and into the scrotum were isolated by sharp dissection. It became apparent that a peritoneal-lined sac of fluid was present. The sac was incised and approximately 30 c.c. of fluid was released under pressure. This fluid had the appearance and consistency of bile-stained, homogenized milk. It was seen that the fluid was coming from the peritoneal cavity through an internal ring 3 mm. in diameter. No testicular tissue was apparent grossly in the sac, although there was one area which appeared to be a small epididymis. No definite cord structures were apparent. Hence, the entire sac was excised at the internal ring, and closure was effected with a purse-string silk suture.

A transverse incision was then made in the right lower quadrant of the abdomen. When the peritoneal cavity was entered, approximately 400 c.c. of fluid similar to that described was released. The fluid was examined by a pathologist,* who confirmed the diagnosis of chyle. The peritoneal surfaces were seen to be smooth and shiny with no apparent reaction. The stomach, duodenum, large bowel, and all abdominal organs were normal. The entire small bowel was somewhat dusky and slightly darker than usual, but there was no evidence of malrotation or internal hernias. The mesentery of the small bowel was moderately edematous, and there was marked distention of lacteals throughout the small bowel, giving the appearance of innumerable white threads. Multiple enlarged mesenteric lymph nodes were present, some of which were 0.5 to 1 cm. in diameter. No source of the chylous leakage could be found. The wounds were closed in layers with interrupted silk sutures. No drain was used.

Postoperatively the abdominal wound drained a small amount of chyle for forty-eight hours and then healed promptly. No further abdominal distention developed and the infant was discharged from the hospital on the tenth postoperative day. The patient was last seen at 5 months of age. At that time he exhibited normal growth and development, with no evidence of recurrence of the ascites.

COMMENT

The cause of acute chylous ascites is undetermined in approximately 50 per cent of the reported cases, but it must be assumed to be rupture of some part of the abdominal lymphatic system, with leakage of chyle into the peritoneal cavity. In other cases, the cause has been reported as mesenteric adenitis, trauma, obstruction of the thoracic duct, intestinal obstruction, strangulated scrotal hernia, and ruptured chylous cyst.

Because of the suddenness of onset, the markedly enlarged mesenteric lymph nodes and distended lacteals, and the absence of any other etiological factor in the present case, it seems reasonable to assume that the inflammatory process in the glands produced a localized obstruction to lymphatic flow, with resultant transudation through intact walls of lacteals or leakage from rupture of channels with congenital weakness of the wall. This seems plausible, in view of the marked distention of the lacteals seen at exploratory laparotomy.

The previously reported incidence, according to age, when the condition is acute, ranged from 1 month to 69 years. The condition occurred predominantly in males. Our patient was a male and was operated upon at 10 days of age.

*Dr. John J. Andujar, Fort Worth, Texas.

The symptoms and signs of an acute abdominal condition are explainable in most cases by peritoneal irritation brought about by the free chyle, just as is seen when there is free blood in the peritoneal cavity. The diagnosis is made by paracentesis or laparotomy. It is interesting to note the absence of leukocytosis or significant fever, and the preoperative impression of acute intestinal obstruction in both our case and the similar one reported by Davis.[1]

Our case was unique in that the presence of irreducible chyle in the scrotum could not be distinguished clinically from an incarcerated inguinal hernia. However, chylocele in association with chronic chylous ascites was described in five of the cases reported by Gribetz and Kanof,[2] and was stated to be reducible in three of these. No mention of reducibility was made in the other two cases. The fact that the tunica vaginalis is partially open during the neonatal period explains this occurrence of chyle in the scrotum.

Treatment consists of surgical correction of any contributing causes found and evacuation of the free chyle. Postoperative drainage of the peritoneal cavity may or may not be used. Because of the small size and delicacy of lymphatic structures in members of this age group, it would not be feasible to repair a leaking lacteal, even if such could be found. For some unexplained reason, the removal of most of the irritant chyle allows the leakage to subside, as evidenced by complete recovery without known recurrence in 25 of the 26 cases of acute chylous ascites reported. The one patient who died was 32 years of age and apparently moribund at the time of operation. If the diagnosis can be made clinically in chronic chylous ascites and no correctable condition is suspected, then paracentesis may be tried initially and may be curative, without resort to surgical exploration. When the condition is acute, however, we feel that laparotomy is the treatment of choice, because a correctable etiological factor may be found in more than 50 per cent of the patients,[3] and complete removal of the fluid apparently results in 100 per cent cure in infants and children when no causative factor can be found.

SUMMARY

The second recorded case of acute chylous ascites in infancy, presumably due to mesenteric lymphadenitis, is presented.

The significant features of this case are described, with a brief discussion of the causation, clinical manifestations, treatment, and prognosis.

No review of the literature is presented because this has been made adequately by other authors.

REFERENCES

1. Davis, N. P.: Chyle Ascites and Other Disturbances of the Chyle System, Pennsylvania M. J. 43: 142-144 (Nov.) 1939.
2. Gribetz, D., and Kanof, A.: Chylous Ascites in Infancy: With Report of a Case With Vitamin A Absorption Studies, Pediatrics 7: 632-641 (May) 1951.
3. Hoffman, W.: Free Chyle in the Acute Abdomen: So-called Chyle Peritonitis, Internat. Abstr. Surg. 98: 209-220 (March) 1954.

INCARCERATED DIAPHRAGMATIC HERNIA WITH GANGRENE OF THE STOMACH

JOSEPH T. SHERIDAN, M.D., CHICAGO, ILL.

THE clinical picture of incarcerated diaphragmatic hernia is unique and so dramatic that the diagnosis should not often be missed. The correct diagnosis has seldom been made before surgery or autopsy, not because the signs and symptoms were misleading, but because incarcerated diaphragmatic hernia was not entertained in the differential diagnosis. Carter and Giuseffi[1] statistically analyzed the clinical findings in a series of forty-three patients and stressed the stereotyped pattern that was so often followed.

In the majority of cases there is a history of previous trauma, and many patients will have noticed premonitory symptoms of sporadic pain in the left upper abdomen or left chest, flatulence, vomiting, constipation, or dyspnea. Symptoms of incarceration almost always begin suddenly and are often related to a sudden increase in intra-abdominal pressure. Coughing, sneezing, vomiting, eating or drinking, straining at stool, or physical exertion may precipitate an attack. The first and most outstanding symptom is pain which is characteristically located in the epigastrium or left upper quadrant of the abdomen or the left lower chest. It is usually severe and colicky. There may be pain in the left shoulder. Soon after the onset of the pain, vomiting begins, and the vomitus may contain blood. Dyspnea, hiccough, and constipation may be present.

The physical findings may be so typical that the correct diagnosis can be made without a roentgenogram. The patient is usually obviously in acute distress and may be in shock. Examination of the chest reveals either dullness or tympany in the lower left chest, absent breath sounds with adventitious sounds such as tinkling, gurgling, or peristaltic sounds in the lower left chest, shift of the heart to the right, and, occasionally, succussion splash. These abnormal chest findings may lead to an impression of primary chest pathology with the result that thoracentesis is attempted. Bloody fluid, even in large amounts, may be aspirated and should be considered further evidence that the hernia is strangulated. The abdomen may be entirely normal. This is especially true when the stomach is the only organ in the hernia. Very careful examination may disclose a slight concavity in the left hypochondrium, and there may be some tenderness in the upper abdomen. Bowel sounds are usually markedly diminished. If intestine is caught in the hernia, there will be the usual signs of bowel obstruction.

X-ray examination of the chest is the most useful tool in making the diagnosis, although careful interpretation is necessary to avoid confusing the findings with other more common conditions. Most important are: (1) simulation of a high diaphragm by the distended herniated viscus; (2) presence of a gas-fluid

Received for publication March 17, 1955.

level above the usual location of the diaphragm; (3) evidence of fluid in the chest; and (4) shift of the heart to the right. The presence of a normal chest roentgenogram does not preclude the diagnosis, however, for the hernia may lie entirely behind the heart shadow. Lateral and decubitus views are particularly useful.

An illustration of almost all these signs and symptoms is found in the case reported herein, with the one difference that the hernia entered the right chest instead of the left. As a result, although the correct diagnosis was briefly considered by one observer, it was dismissed as too improbable, resulting in a delay that was almost disastrous.

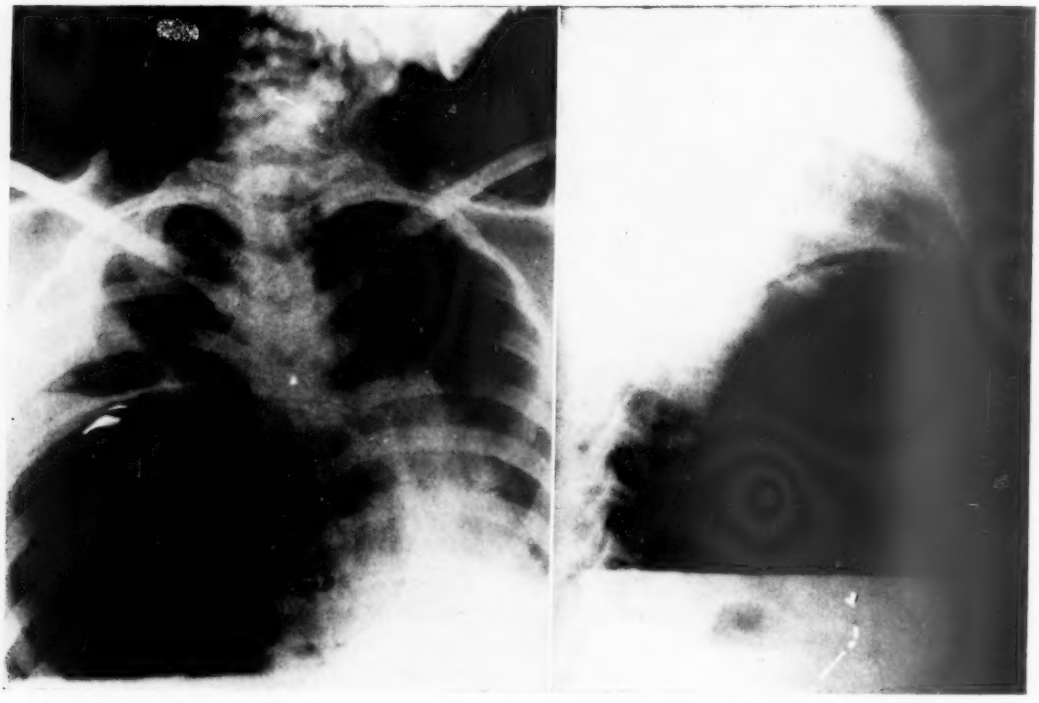


Fig. 1. Fig. 2.

Fig. 1.—Posteroanterior view of the chest on admission showing the large pocket of gas and fluid in the lower right chest. The heart is displaced markedly to the left. (The two light spots in the right mid-chest are artifacts.)

Fig. 2.—Right lateral view of the chest on admission clearly showing the thin line arching over the pocket of gas and liquid.

CASE REPORT

A 36-year-old housewife entered Cook County Hospital, Feb. 17, 1954, complaining of abdominal pain and shortness of breath. She had considered herself in good health until three hours before admission when, while drinking a glass of water, she experienced a sudden urge to vomit. She vomited her lunch, but this gave only partial relief. Following this she felt a fullness in her stomach, a desire but inability to belch, severe epigastric pain radiating girdlewise around the right to her back, and then an increasing shortness of breath. The pain continued with periods of exacerbation and incomplete relief. There was no shoulder pain, no aggravation by breathing, and no relief with movement or change in position. She had never had this or any other significant abdominal pain before and had never had any

symptoms suggestive of peptic ulcer. There was no history of intolerance to fatty or greasy foods and no flatulence or excessive belching. Past history revealed no previous serious illnesses and no operations. She had had chronic sinusitis for many years and recently an acute upper respiratory infection. A chest roentgenogram taken by a mobile tuberculosis survey unit five months earlier was reported as normal. She was a moderately heavy drinker and about one week before admission had been on a heavy drinking bout. She had one child.

Physical examination revealed a well-nourished, well-developed Negro woman in moderate distress. She was quite dyspneic and at times seemed to be seized with sudden pain causing her to sit up in bed. She attempted to vomit but produced only small amounts of mucus. Her pulse was 108, respirations 35, temperature 101.0° F., and blood pressure 100/68. The entire lower right chest was very tympanitic and no breath sounds could be heard there. The heart was shifted to the left. The abdomen was soft and flat, but there was definite tenderness and guarding in the right upper quadrant. Bowel sounds were diminished. Urinalysis was normal. Hemoglobin was 92 per cent and white blood cells 7,550.

Roentgenograms taken on admission revealed a very large, gas-filled space in the lower right chest with a fluid level at the tenth posterior intercostal space. Arching over this space was a thin, curved line which could be seen on both posteroanterior and lateral views, and compressed above this structure was the right lung. A repeat x-ray film taken one hour later showed an impressive rise in the fluid level to the seventh posterior intercostal space.

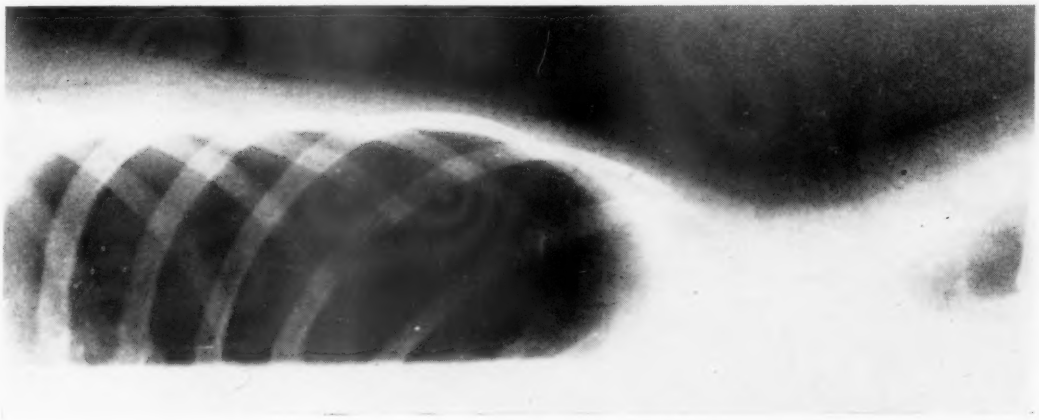


Fig. 3.—Lateral decubitus view of chest and abdomen on admission.

At this time the diagnoses considered were spontaneous pneumothorax and an infected lung cyst. It was felt that the latter best explained all the findings, that there was a "ball valve" mechanism in the connecting peripheral bronchus due to infection from the sinusitis and recent acute upper respiratory infection causing rapid expansion of the cyst. The chest was aspirated with syringe and needle, and two catheters attached to water-seal bottles were inserted into the lower right chest, one anterior and one posterior. It was hoped that one of the tubes would remain inside the lumen of the cyst for drainage even though the cyst might collapse away from the chest wall after being punctured. Gas under pressure was encountered and then about 600 c.c. of thick, foul-smelling, dark-red liquid was aspirated. This "pus" seemed to substantiate the suspicion that the patient had aspirated vomitus while drinking and was thought to be compatible with the diagnosis of infected lung cyst, but pyopneumothorax secondary to perforation of a putrid lung abscess, subphrenic abscess, and amoebic liver abscess with perforation through the diaphragm were also considered. Microscopic examination of the fluid removed revealed no amoebae, but some vegetable fibers were seen. Culture showed *Escherichia coli*, nonhemolytic streptococci, and *Aerobacter aerogenes*.

The severe dyspnea continued, and the blood pressure fell to 70/0. She was given blood, oxygen, antibiotics, and, as the shock became worse, norepinephrine to maintain her blood pressure above 90 systolic. Roentgenograms taken twenty-four hours after admission

showed little change except for pneumothorax in the right chest above the space previously described. Although the tubes in the chest remained patent, none of the foul "pus" was draining through them. Bronchoscopy was performed and a large amount of foamy, mucous secretions was seen, but no other pathology was discovered. She still did not vomit, but continually spat out mucus and saliva. Her nonprotein nitrogen was 96 mg. per cent, creatinine 2.0 mg. per cent, chloride 110 meq. per liter, sodium 144 meq. per liter, potassium 4.4 meq. per liter, and CO_2 combining power 44. An excessive amount of intravenous fluids had been given in maintaining the blood pressure, resulting in an episode of pulmonary edema which disappeared when the fluids were discontinued and tourniquets were temporarily applied to her extremities. When the blood pressure fell again, a more concentrated solution of norepinephrine was used. The temperature remained between 100° to 101° F. By now her condition was desperate.

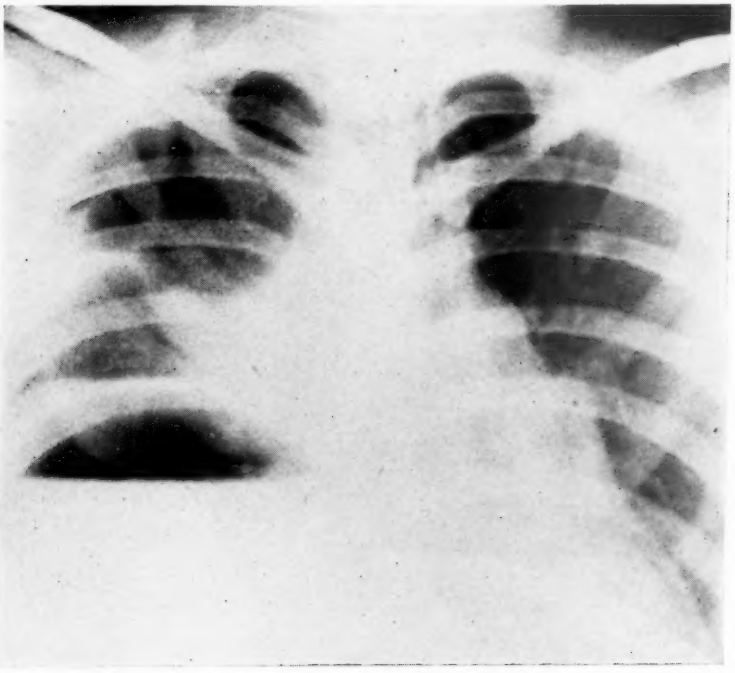


Fig. 4.—Posteroanterior view of the chest taken one hour after admission showing rise in fluid level. A catheter which had been inserted into the chest can be seen.

Because vegetable fibers had been found in the fluid aspirated and because the arched line seen above the space on x-ray film looked so much like diaphragm, it was decided that subphrenic abscess due to perforated viscus represented the most likely diagnosis. Accordingly (forty-eight hours after admission), the right anterior subphrenic space was explored under local anesthesia. A small right subcostal incision was made, and, when no pus was found after an extraperitoneal dissection, the peritoneal cavity was opened. The right lobe of the liver, which was displaced downward 4 to 5 inches below the right costal margin, filled the wound and prevented visualization of other organs. It was determined that the tube through which the "pus" had drained lay entirely above the diaphragm. Since there was no evidence of peritonitis or any other pathology below the diaphragm, and since the patient's condition was so extremely poor, it was decided that further abdominal exploration was not indicated. It seemed unlikely that she would tolerate a thoracotomy, so no further surgery was attempted.

During the succeeding twenty-four hours, further attempts at closed drainage were made by needle aspiration and by insertion of other catheters. A very small amount of foul-smelling gas and thick, dark "pus" was aspirated through a needle, but when a catheter was

inserted at the same site, nothing could be obtained. The dyspnea and shock continued, and more norepinephrine was needed. Because there was continuing evidence of heart failure (râles in both lungs, dyspnea, and tachycardia), she was digitalized. Her temperature rose to 102° F. A swallow of Diodrast was given, and an x-ray film then showed some widening of the lower esophagus and a puddle of Diodrast overlaying the right eleventh rib.

Fig. 5.—Diodrast swallow on the third day just before surgery. The Diodrast can be seen in the slightly dilated lower end of the esophagus and in a puddle on the right below the gas-fluid level. The rounded density on the left is breast shadow.

The patient was again taken to the operating room and the right chest opened through an anterior incision at the fifth intercostal space. A large, cystlike structure filled with the same thick liquid that had been aspirated occupied the entire lower two-thirds of the right chest. The wall was black and obviously gangrenous. This structure proved to be the stomach, which had entered the right chest through a right paraesophageal hernia. About four-fifths of the stomach had protruded into the chest through a defect about 2 inches in diameter which lay just to the right of the normal esophageal hiatus. A few fibers of muscle separated the defect from the esophageal hiatus. The abdomen was opened through an upper midline incision, and the stomach delivered into the abdomen. The esophagus penetrated the diaphragm normally through its hiatus and was viable, but the stomach from the esophagocardiac junction to the distal pyloric antrum was gangrenous. This gangrenous portion of the stomach was resected, and an end-to-end anastomosis performed between the terminal esophagus and the viable pylorus. The defect in the diaphragm was repaired with interrupted 00 silk sutures. The right lung expanded readily to fill the right pleural cavity. The abdomen and chest were closed, leaving two tubes in the chest attached to underwater seals.

Following surgery the patient's condition was still extremely poor, and for two days norepinephrine was needed to keep the blood pressure above shock level. She was given

transfusions of whole blood to equal blood lost during surgery. The nonprotein nitrogen rose to 138 mg. per cent, but the electrolytes were kept within normal limits by careful attention to fluid balance. When the blood pressure returned to normal, the urine output was adequate. The nonprotein nitrogen slowly returned to normal over the next two weeks. The hematocrit returned to 45 per cent after transfusions. She was allowed small sips of water on the third postoperative day, and her oral intake was very slowly increased in amount and consistency thereafter. This proceeded without mishap, and by the time she left the hospital, she was eating three or four meals of general hospital diet supplemented with one quart of high protein liquid mixture and was gaining weight. All of her wounds healed without infection. Postoperative chest roentgenograms showed marked haziness involving the entire right lower lung field. This was interpreted as pleural thickening and fluid with residual atelectasis and pneumonitis. Repeated attempts at thoracentesis produced no significant amount of fluid. The opaque areas gradually shrank until only a small pocket in the right anterior chest remained. Needle aspiration again produced nothing, and subsequent roentgenograms showed that this too was resolving. When the patient left the hospital two months and ten days after admission, she had no symptoms. In a telephone conversation one year later, she complained only of being unable to eat large amounts at one time.

As mentioned before, the diagnosis in this case seems disturbingly obvious in retrospect. Although the history contained no premonitory symptoms, the sudden onset, the character of the pain, the ineffectual vomiting, and the desire to belch were certainly characteristic. By direct questioning later, when we knew the diagnosis, it was learned that the patient had noticed dyspnea after meals for several months before admission. The severe vascular collapse, the dyspnea, and the abnormal chest findings directed our attention to the thorax. We were misled by the minimal abdominal findings, and later, when the abdomen was opened, by the absence of peritonitis. The reward of a thorough exploration at every laparotomy is well shown. Had we discovered that the stomach was not in the abdominal cavity, twenty-four hours would have been saved. Having become so engrossed in pulmonary pathology, we were guilty of several glaring oversights. Although the patient was not vomiting in the hospital, she continually spat out all her saliva (yet did not complain of difficulty in swallowing). A Levine tube was not inserted since she had stopped vomiting; we did not suspect abdominal pathology, and we did not want to do anything that would interfere with her breathing. A Levine tube would have aided greatly in the diagnosis. Although the roentgenograms fulfill all the criteria given here, a strangulated diaphragmatic hernia is certainly not the first thing one thinks of in looking at them. We did overlook the fact that on none of the films is the stomach gas bubble present beneath the left diaphragm. Observance of such a detail would have led to an earlier Diodrast swallow study. Having misinterpreted as pus the foul fluid we had aspirated, we were both puzzled and vexed that nothing came through the tubes we inserted to drain the "abscess." When the gangrenous stomach wall was examined later at surgery, it was found that the puncture holes of the needles had sealed over, while the blunt catheters had pushed the stomach wall away without piercing it. The patient probably owes her life to the fact that we were not able to produce a leak in her stomach that would have grossly contaminated her pleural cavity.

SUMMARY

A case of gangrene of the stomach within the right chest due to an incarcerated paraesophageal diaphragmatic hernia with successful surgical treatment is presented. The diagnosis of incarcerated diaphragmatic hernia is discussed.

Acknowledgment is made to Dr. Richard Bodie of Aurora, Illinois, who performed the gastric resection and repair of the hernia, and to Dr. James Kane of Chicago, Illinois, who assisted in the care of the patient.

REFERENCES

1. Carter, B. N., and Guiseffi, J.: Strangulated Diaphragmatic Hernia, Ann. Surg. **128**: 210, 1948.
2. Deaner, S., McMenemey, W. H., and Smith, S. M.: Hemothorax Due to Strangulated Diaphragmatic Hernia, Brit. M. J. **1**: 72, 1943.
3. Gants, R. T.: Diaphragmatic Hernia, Ann. Surg. **139**: 166, 1954.
4. Hoffman, W., Levy, M. L., Sole, E., and Lewitan, A.: Strangulated Diaphragmatic Hernia With Gangrene of the Stomach, Arch. Surg. **69**: 125, 1954.
5. Hurley, G. A. P.: Strangulated Hiatus Hernia, Ann. Surg. **138**: 262, 1953.
6. Kushlan, S. D.: Strangulated Right Diaphragmatic Hernia With Surgical Cure, Gastroenterology **18**: 466, 1951.
7. Pearson, S.: Strangulated Diaphragmatic Hernia, Arch. Surg. **66**: 155, 1953.

MINIMAL FOCAL PANCREATITIS: A CAUSE OF INCAPACITATING ABDOMINAL PAIN

JOSEPH A. BUCKWALTER, M.D., IOWA CITY, IOWA

(Department of Surgery, College of Medicine, State University of Iowa)

THE growing clinical significance of disorders of the pancreas is reflected by the increasingly prominent place considerations of this organ occupy in the medical literature.[1, 2] Advances which have made surgical treatment of pancreatic lesions feasible are perhaps chiefly responsible. However, there remain areas in which information is incomplete and therapy unsatisfactory. The cause of acute and chronic pancreatitis is poorly understood. It seems probable that they represent stages in a disorder which characteristically progresses from an acute process with remissions and exacerbations to a chronic one with irreversible pathologic changes which result in permanent physiologic disturbances.[3] The symptoms and signs of the acute and the chronic phase of the disease have been well delineated.[4, 5] The mechanisms involved in their production have been suggested by various authors.[6, 7] The deposition of calcium with the formation of calculi scattered diffusely through a large, destroyed pancreas would seem to be one of the end results of this slowly progressing inflammatory process.

The case reported is of interest because of the unusual clinical features, the minimal pathologic changes, and the failure on review of the relevant literature to find recorded a similar one.

CASE HISTORY

B. D. H., a 75-year-old white man, stated that he had been entirely well until the onset of his present illness ten days before his first visit to the University Hospitals. As was his custom, he had retired about 10 P.M. that evening. There had been no suggestion in the days and weeks before that he was other than in his usual state of good health. He was awakened at 3 A.M. by severe, penetrating pain in the left hypochondrium. This pain persisted in its very severe form for more than one hour. At that time his physician was called, and the pain was somewhat relieved by the administration of aspirin. There was no nausea, vomiting, chills, temperature elevation, or abdominal tenderness. The pain persisted for the remainder of the night, becoming less severe. When the patient dressed in the morning, the pain was gone; there were no anorexia or other symptoms. During the day he enjoyed his usual state of health and was able to carry on his customary activities.

Similar attacks of severe paroxysmal upper left quadrant pain occurred at almost the same time each night until his visit to the University Hospitals. On some occasions the pain would be somewhat relieved by taking aspirin and codeine; on others, not. The patient volunteered the information that, at times, getting up out of bed, walking, or sitting in a chair in an upright position seemed to relieve his symptoms. At no time did he have the pain during the waking hours of the day. There continued to be no gastrointestinal tract symptoms. On several examinations by his family physician, there was no abdominal tenderness or any other positive physical signs.

This patient at the time of the onset of his illness was active in his capacity at the bank. His responsibilities there required seven or eight hours daily. In addition, he carried

Received for publication March 31, 1955.

on an active social life in the community. A physician's attention had been unnecessary since his last visit to the University Hospitals seven years before. At that time a transurethral resection had been performed to relieve bladder neck obstruction due to hyperplasia of the prostate. During this interval he had been entirely well. The only other significant point in his past medical history was the reference to a fall some twenty years before which had resulted in a vague back injury. Pain in the upper lumbar region had limited his activities for about a week. No roentgenograms were obtained and there were no subsequent difficulties. The patient categorically denied a previous episode of abdominal pain in any sense similar to the current one. Careful interrogation failed to disclose the history of any illnesses which might have represented cholecystitis, choledocholithiasis, pancreatitis, diverticulitis, appendicitis, duodenal ulcer, or gastritis.

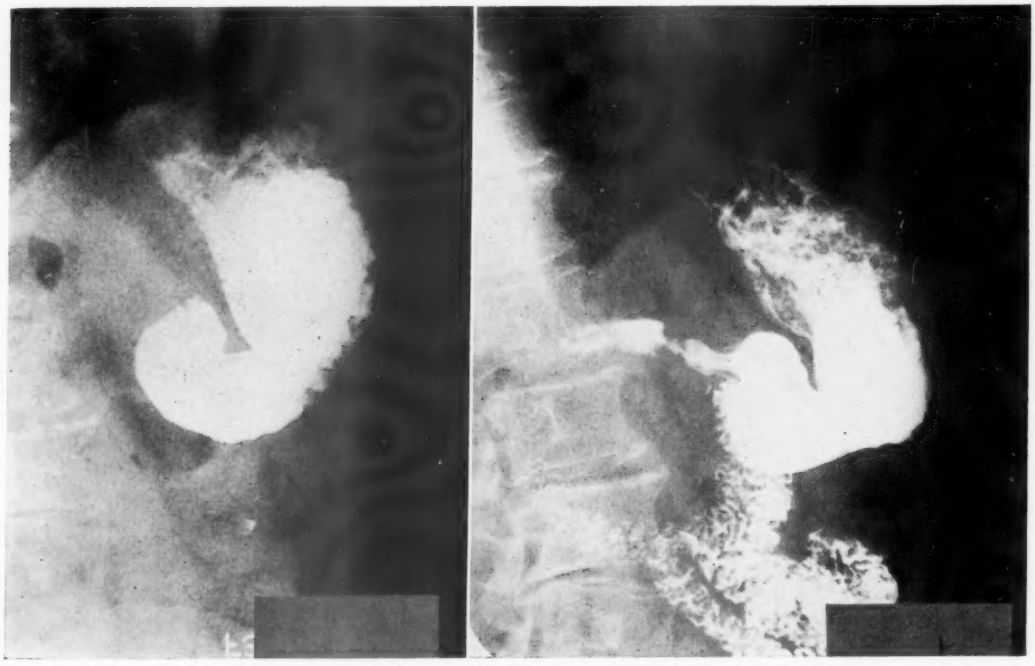


Fig. 1. Fig. 2.

Fig. 1.—Persistent pylorospasm occurring during an episode of abdominal pain.
Fig. 2.—Several days later there was no pylorospasm in the absence of the abdominal pain.

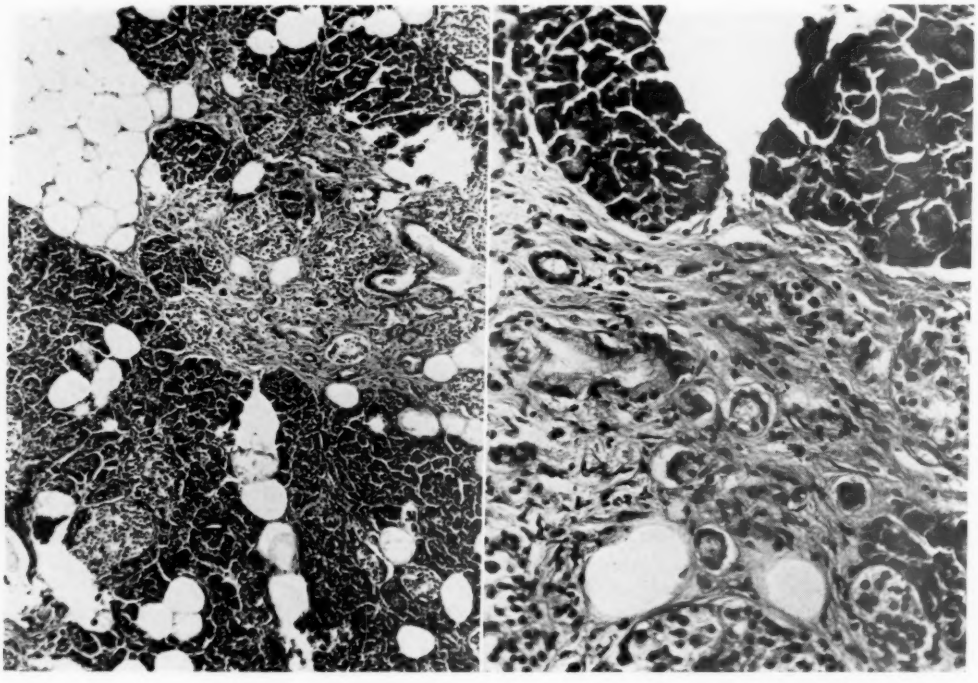
Physical examination revealed a healthy, tall, lean, well-muscled, well-nourished white man who did not appear to be physiologically more than 65 years of age. The patient was cooperative and appeared to have an entirely objective point of view in his desire to find the cause for his present complaint. The blood pressure was 180/100 mm. Hg. A left indirect reducible hernia and a diffusely enlarged soft prostate were the significant positive physical findings. There were no signs of peritoneal irritation.

The red blood count was 4,700,000 cu. ml.; white blood count 7,800 cu. ml.; and hemoglobin 13.5 Gm. per 100 c.c. Gross and microscopic urinalysis was normal. Two blood amylase determinations made during the time the patient was experiencing pain showed 71 and 40 units; urine diastase was 145 units. The blood van den Bergh test was 1.0 mg. per cent; cephalin flocculation for 24 hours, 1+, for 48 hours, 1+.

A photoroentgenogram of the chest revealed no abnormalities. On the day that an upper gastrointestinal barium study was to be performed, the patient was experiencing abdominal pain. Pylorospasm prevented the passage of the barium into the duodenum (Fig. 1). Repeated a few days later with the patient free from pain, there was no pylorospasm (Fig. 2).

The esophagus, hiatus, stomach, and duodenum appeared to be normal. Motor meal studies indicated no abnormality of the small intestine. Intravenous pyelograms showed prompt bilateral function. A left retrograde pyelogram revealed a normal ureter and renal pelvis. A sigmoidoscope was readily passed to 25 cm. with no abnormalities being noted. A barium enema showed the presence of diverticulosis of the sigmoid colon with some spasm.

During the period of more than two weeks that he was under observation, the patient continued to have almost nightly attacks of the same type of pain which has been described. Repeated examinations during these periods failed to disclose any additional significant physical findings. At no time did the patient have any signs of peritoneal irritation. There continued to be no disturbance in gastrointestinal function. With the completion of the work-up, the findings were frankly discussed with the patient. An exploratory laparotomy was advised with the admission that there was a very real possibility that it would prove negative. It was felt that the symptoms were sufficiently suggestive of carcinoma of the body or tail of the pancreas that an operation was indicated. The patient elected to defer operation at this time.



A. *B.*

Fig. 3.—Minimal pancreatitis (*A*, ×75; *B*, ×150; reduced ½).

Two weeks later he returned to the hospital stating that the attacks of severe upper abdominal pain had persisted and that he was now prepared to accept operation. Exploratory laparotomy was performed one month after the onset of the illness. Abnormalities were limited to the pancreas. In the body just to the left of the superior mesenteric vessels, the pancreas appeared to be swollen and quite hard. The area involved was about 3 to 4 cm. in diameter. The remainder of the organ including the tail did not appear to be abnormal. The gross impression was carcinoma of the pancreas. Multiple biopsies were obtained and frozen sections made. These revealed changes compatible with a mild chronic pancreatitis. The subsequent fixed tissue sections confirmed these findings (Fig. 3, *A* and *B*). The islet tissue was well preserved. Interlobular fibrosis was noted with foci of chronic inflammatory cells. There was no evidence of an acute inflammatory process or neoplasia. The pancreas was transected just to the left of the superior mesenteric vessels well to the right of the

gross lesion. The tail and the part of the body to be resected were readily mobilized from their retroperitoneal attachments and removed (Fig. 4). Gross evidence of a peripancreatic inflammatory process and fat necrosis was lacking.

The patient's postoperative course was uneventful. He was discharged from the hospital seven days following operation. At this time his wound was healing well, he was taking a general diet, and there had been no return of the preoperative upper abdominal pain. No suggestion of exocrine or endocrine pancreatic deficiency was present. In the two years since the operation, the patient has remained entirely well. He has carried on as before the onset of his illness and has had no return of his preoperative pain.

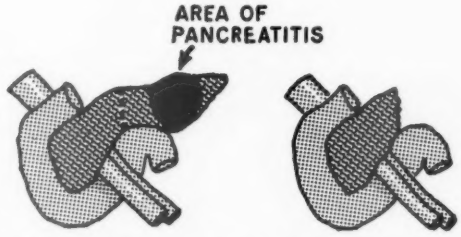


Fig. 4.—Diagram showing the position of the lesion and the part of the pancreas excised.

COMMENT

The preoperative diagnosis of carcinoma of the body or tail of the pancreas was based upon positive and negative evidence. Paroxysmal abdominal pain which radiates to the back, worse in the supine position, is one of the most constant and significant symptoms associated with this lesion.[8, 9] Pylorospasm, more often associated with pancreatitis, is an indication of the reflex motor disturbances of the upper gastrointestinal tract which frequently accompany the pain of pancreatitis.[7] The absence of anorexia, weight loss, jaundice, and other indications of gastrointestinal tract dysfunction did not weigh heavily against the preoperative diagnosis. Similarly, the negative physical findings, laboratory, and x-ray studies, by excluding other possible diagnoses, seemingly increased the probability that the presumptive diagnosis was correct. The most important point against this diagnosis was the short history in an entirely well individual.

The gross lesion found at operation was more impressive than the inflammatory changes noted in the microscopic sections. The prompt relief of the incapacitating pain following surgery, which has now persisted for more than two years, would seem to unquestionably implicate the minimal lesion which was excised.

The etiology and pathogenesis of this lesion remain obscure. Current concepts do not provide us with a plausible explanation. There was every reason to accept this patient's denial of any previous episode which could be construed as acute pancreatitis, as a matter of fact. Vascular thrombosis with a small area of infarction, blood-borne focal infection, or local release of activated enzyme through a damaged duct are suggested as possible etiological factors. Whether the described back injury had any relationship to this lesion must remain a matter for speculation.

SUMMARY

The case history of a patient with incapacitating abdominal pain relieved by excision of a minimal inflammatory lesion in the body of pancreas has been reported. Exploratory laparotomy was indicated because of our inability to rule out carcinoma of the body or tail of the pancreas.

REFERENCES

1. Cattell, R. B., and Warren, K. W.: Surgery of the Pancreas, Philadelphia, 1953, W. B. Saunders Company.
2. Elman, R., and Wheat, M. W.: The Pancreas: Contributions of Clinical Interest in 1952, Gastroenterology **26**: 149, 1954.
3. Probstein, J. G.: Pancreatitis: Current Concepts, J. Internat. Coll. Surgeons **18**: 489, 1952.
4. Paxton, J. R., and Payne, J. H: Acute Pancreatitis: A Statistical Review of 307 Established Cases of Acute Pancreatitis, Surg., Gynec. & Obst. **86**: 69, 1948.
5. Comfort, M. W., Gambill, E. E., and Baggenstoss, A. H.: Chronic Relapsing Pancreatitis; Study of 29 Cases Without Associated Disease of Biliary or Gastrointestinal Tract, Gastroenterology **6**: 239-285 (Apr.); 376-408 (May) 1946.
6. Ray, B. S., and Neill, C. L.: Abdominal Visceral Sensation in Man, Ann. Surg. **126**: 709, 1947.
7. Snell, A. M., and Comfort, M. W.: The Incidence and Diagnosis of Pancreatic Lithiasis, Am. J. Digest. Dis. **8**: 237, 1941.
8. Smith, B. K., and Albright, E. C.: Carcinoma of the Body and Tail of the Pancreas, Ann. Int. Med. **36**: 90, 1952.
9. Brown, R. K.: The Early Diagnosis of Cancer of the Pancreas Based on the Clinical and Pathological Study of One Hundred Autopsied Cases, Am. J. Med. Sci. **223**: 349, 1952.

THE TIKHOR-LINBERG RESECTION OF SHOULDER GIRDLE

Case Report

George T. Pack, M.D., and James C. Baldwin, M.D., New York, N. Y.

(From the Pack Medical Foundation)

THE Tikhor-Linberg operation for resection of the shoulder girdle encompasses total scapulectomy, partial or complete excision of the clavicle, and resection of the head and neck of the humerus with preservation of the arm, brachial plexus, subclavian artery, and vein.

The operation was first planned by the Russian surgeon Tikhor and applied afterward by Bauman and Linberg at the turn of the century. The purposes of the operation were threefold: (a) as a preferable method of treating certain tumors of the shoulder when the patient refused amputation; (b) for certain cancers or even unsual destructive inflammatory or traumatic diseases when the neurovascular bundle was intact; and (c) the preservation of an upper extremity capable of limited, but valuable, function.

Technique of the Operation.—A racket incision is made along the anterior surface of the clavicle and extended laterally over the deltoid muscle at a distance of 1 to 3 inches down the arm. The removal of the clavicle allows an adequate exposure of the subclavian vessels and brachial plexus ensuring their careful preservation. It also makes possible the ligation of the blood vessels prior to resection of the scapula and head of the humerus. Division of the pectoral muscles permits a meticulous dissection of the brachial plexus and subclavian vessels. The deltoid muscle is transected at its attachments to the clavicle, acromion process, and spine of the scapula. The transverse cervical and suprascapular arteries are located, ligated, and severed. The long and short heads of the triceps muscle are severed at their insertions. The humerus is then transected at or below the surgical neck. The posterior incision is carried over the scapula, the trapezius muscle is removed from the spine of the scapula, the levator scapulae rhomboid major and minor muscles are removed medially, and the scapula is raised. The teres major muscle and the long head of the triceps are resected and the specimen is removed. The biceps and triceps muscles are sutured anteriorly and posteriorly to the intercostal muscles and anchored to the periosteum of the ribs. The remaining soft tissues and skin are apposed and sutured in the conventional manner.

Scapulectomy alone, as described by Syme in 1864, is still performed occasionally, and then usually for primary bone tumors such as chondrosarcoma. de Nancrede, in a comprehensive review written in 1909, commented on the inferior end results of scapulectomy for malignant tumors. Metastatic cancer in the scapula is infrequent (5 in 5,734 cases reported by Geschickter and Maseritz, and a solitary metastasis almost never occurs. Radiation therapy is usually preferable to surgical extirpation for these lesions. Resection of the head of

Received for publication March 31, 1955.

the humerus, because of its proximity and functional relationship to this region, causes no great postoperative problems and the original disease, cancer or otherwise, may involve this bone segment itself.

The rationale in the choice of this procedure is not to supplant the recognized procedures for surgical illnesses of this region, such as the more conservative total scapulectomy previously mentioned and the conventional interscapulothoracic amputation, but is exemplified by certain rare instances such as recited in the present case report. There has been very little discussion of this procedure in medical literature because of the limitations of its use. A study of published case reports of this operation lends support to the belief that it is chiefly palliative, because the patients who have not died have often required subsequent and more radical operations for recurrences. The only alternative for the Tikhor-Linberg operation is the interscapulothoracic amputation.*

Some of the lesions involving the shoulder girdle which might furnish indications for the Tikhor-Linberg shoulder girdle resection are:

A. *Inflammatory, suppurative diseases,* associated with massive bone necrosis, multiple fistulas, joint destruction, sepsis, and so on. Since the introduction of antibiotics, this indication must have become quite infrequent.

B. *Metastases to shoulder girdle.* Crippling destructive metastases to bone constituting postirradiation failures. If the patient would otherwise be anticipated to have a comfortable life expectancy, this operation might well be used more frequently as a palliative measure.

C. *Bone tumors.* Osteogenic sarcoma and chondrosarcoma of scapula invading soft tissues and joint components, and unsuitable for scapulectomy. We cannot conceive of a situation in which the radiosensitive endothelial myeloma would qualify for an operation of this character.

D. *Synovial sarcoma (malignant synovioma).* Although this tumor is not of frequent occurrence around the shoulder joint, it theoretically would be the most suitable of all soft part sarcomas for this specific operation.

E. *Liposarcoma.* The operation would generally be contraindicated for this tumor. Liposarcomas are most frequent in the shoulder and pelvic girdles, but seldom compromise bone; they have some degree of radiosensitivity and require extensive sacrifice of soft tissues.

F. *Rhabdomyosarcoma.* This tumor would seldom be suitable for the Tikhor-Linberg operation because it usually does not involve bone; it may be multicentric in origin, and experience has shown that a fundamental law governing its surgical treatment is to excise all of the muscle groups involved from origin to insertion or to amputate proximal to the origin of the muscle groups.

G. *Fibrosarcoma.* This tumor is commonly diffuse and invasive and may produce a local condition in the shoulder girdle which will tempt the surgeon to perform the Tikhor-Linberg operation and preserve the arm. Opposed to this consideration is the pernicious tendency of this sarcoma to extend widely up and down along the fascial planes far beyond the scope of the usual conservative operations.

*A separate report by Dr. Pack covering the indications, limitations, and end results of 88 interscapulothoracic amputations for tumors is in press.

H. *Malignant neurilemmoma.* A tumor, whch may invade bone or erode it by continuity; it can be pseudoencapsulated or invasive. Two hazards, namely, involvement of the brachial plexus and the predisposition for recurrence proximally along the nerve trunks may render it unsuitable for this operation.

I. *Deeply invasive cancers of the skin.* Epidermoid carcinomas of the skin of the shoulder may involve the bones and shoulder joint. As in the present case report, they may have recurred after attempts at radiation therapy and are sometimes associated with radiation fibrosis and ulceration, necrosis, suppuration, fixation of joint, intractable pain, and disability.

At the completion of the operation, the lower arm, forearm, and hand are intact and the functions of the forearm and hand are largely retained but, of course, shoulder and upper arm movements are no longer possible. The forearm maintains its function and utility completely, with the carrying angle being maintained without difficulty. As previously stated, the boneless upper portion of the arm is fixed by suturing it to the chest wall; after complete healing, followed by training of the arm, some carrying may be done but, of necessity, with limitation as to weight in comparison with the normal arm. To use this arm above the horizontal, it is necessary to use the other arm for support, but it can be raised for such functions as combing the hair, and so on, without discomfort. The cosmetic results are such that a good tailor or the use of a properly fitting prosthesis can build up the shoulder for the proper wearing of clothes.

The actual indications for the Tikhor-Linberg operation must be quite infrequent as the following case report represents the only time it has been employed during a considerable experience with major amputations, that is, interscapulothoracic amputation, 88 cases; hip joint disarticulation, 94 cases; interilioabdominal amputation (hemipelvectomy), 46 cases (G. T. P.)

CASE REPORT

F. H., a 58-year-old married woman, was admitted to the hospital on Dec. 5, 1953. Her history dated back approximately twenty years when a skin cancer on the posterior right shoulder was treated by x-ray therapy. Following the x-ray treatment, the patient had severe x-ray dermatitis which evolved into chronic radiation dermatitis. Two years prior to admission a nodule was found in the irradiated skin over the right scapula. A biopsy was reported as squamous-cell carcinoma. Radiation therapy was again administered. A deep ulceration resulted which gradually expanded in surface dimension and deepened to invade and expose the bones of the shoulder joint. For four months prior to admisson the pain had been excruciating and the shoulder joint was frozen. Demerol was required every two hours for the relief of pain.

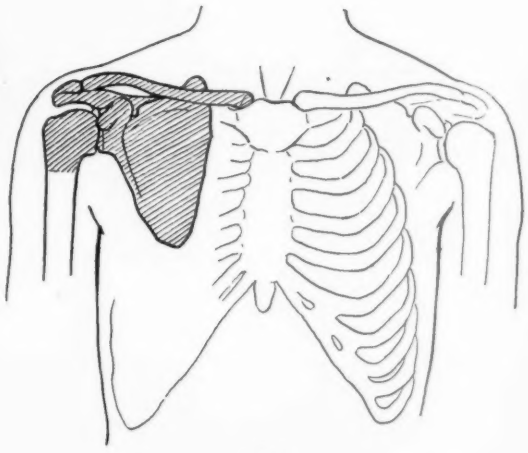
On physical examination the patient was seen to be a pallid, chronically ill woman, constantly guarding the right shoulder. A purple radiation pigmentation with subdermal fibrosis, atrophy, and telangiectasia was found on the posterior and superior aspects of the right shoulder. An ulcer measuring 3 inches wide by ½ inch deep was seen in the center of the pigmented area. The base of the ulcer was typically green and sloughing. The shoulder was immobile; any attempt to elicit motion in the shoulder caused extreme pain.

A diagnosis of recurrent squamous-cell carcinoma occurring in radiation dermatitis and associated with bone invasion and suppurative arthritis of the shoulder was made. Roentgenograms of the shoulder showed bone destruction of the superior margin of the right scapula.

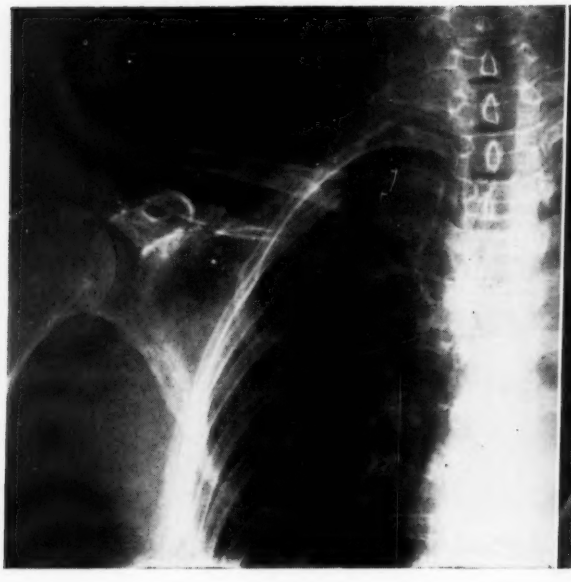
On Dec. 1, 1953, the Tikhor-Linberg procedure was performed. On Jan. 8, and Jan. 22, 1954, débridement of the wound, transposition of skin flaps, and split-thickness skin grafts

were accomplished. The patient was discharged from the hospital on February 4, with the wound healed, enjoying surcease of pain and moving the lower arm freely. On Dec. 23, 1954, when last seen, the wound was completely healed. The patient could raise her right arm for combing her hair, by using her left arm for support. She could use the arm and hand for eating and performed the necessary chores in the routine of a housewife.

A.



B.



C. *D.*

Fig. 1.—*A*, Deformity after resection of clavicle, scapula, head of humerus, and shoulder joint.
B, Schematic outline to show osseous structures sacrificed in the operation.
C, Preoperative roentgenogram illustrating destruction of scapula.
D, Postoperative roentgenogram.

The pathologic report was primary squamous-cell carcinoma of the skin; radiation dermatitis; radionecrosis of the soft tissues; postirradiation osteitis of the scapula; chronic fibrosing osteitis of the humerus; and suppurative arthritis of the shoulder joint.

REFERENCES

1. Abbott, LeR. C., and Lucas, D. B.: The Function of the Clavicle; Its Surgical Significance, Ann. Surg. **140**: 583-599, 1954.
2. Bauman, P. K.: Resection of the Upper Extremity in the Region of the Shoulder Joint, Khirurg. Arkh. Velyaminova, S.-Peterb. **30**: 145-149, 1914.
3. Berger, P.: Amputation du membre supérieur dans la contiguité du tronc (désarticulation de l'omoplate), Bull. et mém. Soc. nat. chir. **9**: 656, 1883.
4. Geschickter, C. F., and Maseritz, I. H.: Skeletal Metastasis in Cancer, J. Bone & Joint Surg. **21**: 314-322, 1939.
5. Ghormley, R. K., and Valls, J. E.: Metastasis to Bone From Carcinoma of Gastrointestinal Tract, J. Bone & Joint Surg. **21**: 74-78, 1939.
6. de Nancrede, C. B. G.: End-results After Total Excision of the Scapula for Sarcoma, Ann. Surg. **50**: 1-22, 1909.
7. Pack, G. T., McNeer, G., and Coley, B. L.: Interscapulothoracic Amputation for Malignant Tumors of the Upper Extremity; a Report of Thirty-one Consecutive Cases, Surg., Gynec. & Obst. **74**: 161-175, 1942.
8. Ryerson, E. W.: Excision of Scapula; Report of Case With Excellent Functional Result, J. A. M. A. **113**: 1958-1960, 1939.
9. Syme, J.: Excision of the Scapula, Edinburgh, 1864, Edmonston & Douglas.
10. Tikhor, P. T.: Tumor Studies, Russia, 1900.
11. Wakeley, C. P. G.: A Case of Sarcoma in the Infraspinatus Muscle, Brit. J. Surg. **26**: 439-442, 1938.

SICKLE-CELL ANEMIA, SALMONELLA ENTERITIDIS OSTEOMYELITIS, AND REMOTE POSTOPERATIVE WOUND ABSCESS

Report of a Case

Ira S. Goldenberg, M.D., New Haven, Conn.

(From the Department of Surgery, Yale University School of Medicine)

THE Salmonella group of organisms is among the less common of the varied bacteria which cause osteomyelitis. Bone infection as a complication of Salmonella-caused disease is also rare, the occurrence rate being about 0.85 per cent of cases of typhoid fever and 0.2 per cent of all other types of Salmonella.[1] *Salmonella enteritidis* isolation from 1,000 cases of Salmonella infection reported by Seligman and associates[2] (excluding *S. typhi*) was only 0.3 per cent, which included one case of osteomyelitis. Thus, it can be seen readily that *S. enteriditis* osteomyelitis is indeed an unusual lesion.

The occurrence of Salmonella osteomyelitis in patients with sickle-cell anemia has been noted previously by Smith,[3] and he believed that more than a casual relationship exists between the two entities. Smith emphasized that both disease processes are marked by thrombosis and infarction. The sickle-cell anemia probably encourages the development of osteomyelitis in the presence of the well-known lowered resistance of these patients, in general, to infection.

Splenic abscesses are not uncommon in patients with septicemia and operative intervention is frequently necessary. It is not uncommon, further, for sterile or infected abscesses to develop at the wound site of any surgical drainage during the postoperative period. It is unusual, however, for a secondary wound abscess to develop in a clinically well patient months after drainage and adequate antibiotic therapy and to obtain a specific positive culture from the pus of such an abscess.

The present report is that of a child who combined all of these—sickle-cell anemia, *S. enteritidis* osteomyelitis, and remote postoperative wound infection positive on culture for *S. enteriditis* following splenectomy for abscess.

CASE REPORT

An 11-year-old boy of Sicilian parents was admitted to the Yale-New Haven Medical Center for the third time on Nov. 29, 1953. Thalassemia major with sickling had been demonstrated at age 7. Intermittent attacks of myalgia had been present since age 4, and hospitalization at age 7, and again at age 8, was required for therapy of acute hemolytic crises. Since the second admission the patient had done well with ferrous sulfate medication, having had only occasional minimal hemolytic episodes. Approximately three days prior to the third admission, the boy noted the onset of pain in both legs and the lumbar region. Temperature elevation to 105° F. (rectal) was noted on the day of admission, accompanied by pain in the left shoulder. Some dysuria was present during the days immediately prior to hospital entry, and the patient's mother believed that his urine was "golden yellow" the day before hospitalization. Physical examination at the time of admission revealed an

Received for publication April 4, 1955.

acutely ill child complaining of pain in the thighs. Slightly icteric sclerae were noted. Examination of the heart revealed the point of maximum impulse to be at the left anterior axillary line, and a Grade 2 systolic murmur was heard loudest at the pulmonic valve area. The spleen was palpable 2 cm. below the costal margin while the liver edge was felt 4 cm. below the rib margin. Bilateral costovertebral angle tenderness was present. Both anterior thighs were slightly tender to palpation. Admission hemogram included hemoglobin of 10.0 Gm. per cent, hematocrit of 34 per cent, red cell count of 4.34 million, white cell count of 19,800 with 94 per cent polymorphonuclear leukocytes. Marked poikilocytosis, anisocytosis, and hypochromia were present. Urine examination was remarkable only for 1 plus albumin. A smear of the urinary sediment was negative for organisms. Blood cultures drawn at this time were reported as positive for gram-negative bacilli. The day after admission these were identified as *S. enteritidis* sensitive to Terramycin, Aureomycin, streptomycin, and Chloromycetin. Liver function tests at the time of admission were direct serum bilirubin 7.16 mg. per cent, total serum bilirubin 14.08 mg. per cent, cephalin flocculation 4 plus, urine bile 4 plus, and a serum alkaline phosphatase of 11.2 mg. per cent.

The patient was started on Terramycin and streptomycin, and 300 ml. of whole blood was administered. Following the transfusion, the patient became markedly icteric and the hemoglobin fell to 3.0 Gm. per cent. At this time, the spleen was noted to be larger than on admission and was now tender. Six hundred milliliters of whole blood was given after the hemolytic episode and the hemoglobin rose to 7.4 Gm. per cent. Four hundred milliliters of packed red cells was given the following day, and Chloromycetin was added to the therapy because the patient remained markedly febrile and repeat blood cultures were positive for *S. enteritidis*. Serum bilirubin rose to 24 mg. per cent (direct) and 41.8 mg. per cent (indirect) four days after the postadmission hemolytic crisis. Enteric serum agglutinins at this time revealed typhoid O—complete 1/160; typhoid H—complete 1/40; paratyphoid A—complete 1/80; and paratyphoid B—trace 1/80.

Cortisone was started on the seventh hospital day and the patient improved markedly after three days of the drug (the cortisone was continued for eleven days, being supplemented by ACTH during the last four days as the cortisone dosage was decreased prior to discontinuing it). Stool culture on the seventh day revealed pure hemolytic *Staphylococcus aureus*, and the Terramycin was discontinued. Blood transfusions, including 250 ml. of whole blood and 250 ml. of packed red cells, were given at this time to bolster a decreasing hemoglobin level. The child continued to improve except for a low-grade fever, and gastroduodenal and gall bladder roentgenograms made at this time were remarkable only for nonvisualization of the gall bladder. Blood cultures at this time did not show any organisms, and a sickling preparation revealed 75 per cent sickling after twenty-two hours. Electrocardiogram was normal.

Weakness of the right arm and hand was noted at this time and was felt to be on the basis of disuse, and physiotherapy was started. Agglutination studies now were typhoid O—complete 1/320; typhoid H—complete 1/160; paratyphoid A—negative; paratyphoid B—trace 1/20. Because of a depressed platelet and white blood cell count, Chloromycetin was discontinued after twenty days of administration. An additional 500 ml. of packed red cells was given on the thirty-third hospital day because of a hemoglobin level of 6.7 Gm. per cent. Streptomycin was discontinued after twenty-seven days of administration.

The patient continued to be febrile without apparent cause during this period. Parenteral fluids were given intermittently to supplement the low oral intake of food and fluids. On the thirty-seventh hospital day Terramycin therapy was started again empirically. Some left flank pain developed and physical examination revealed decreased breath sounds at the left pulmonary base without diaphragmatic movement, and left upper quadrant abdominal tenderness was present. A chest roentgenogram was consistent with left pleural effusion, and subdiaphragmatic abscess was suggested as a possibility for the continued fever. Exploratory laparotomy was advised, and on the forty-fifth hospital day splenectomy and drainage of a splenic abscess were accomplished. Culture of the abscess material revealed *S.*

enteritidis. Streptomycin and penicillin were given immediately after operation, but were discontinued and replaced by Terramycin after the organism had been identified. Postoperatively the patient did well.

Roentgenograms of the right leg (Fig. 1), which were taken immediately prior to operation because of persistent pain in the lower leg, showed an area of destruction in the mid-portion of the shaft of the fibula, possibly an abscess. A postoperative chest roentgenogram showed, as an incidental finding, a focus of osteomyelitis in the left humerus, a place

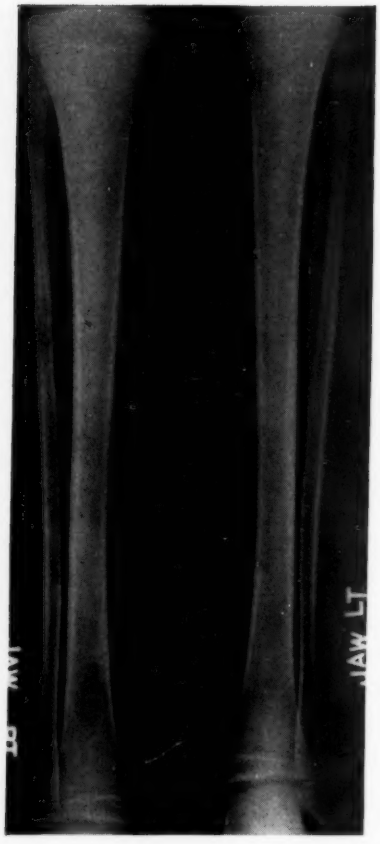


Fig. 1.

Fig. 2.

Fig. 1.—A solitary, localized destructive process is seen in the mid-portion of the shaft of the right fibula slightly above the mid-point. The destruction is symmetrical about the cortex. There is slight periosteal new bone formation.

Fig. 2.—An extensive destructive process is seen in the humerus which extends from the epiphyseal line above, to the supracondylar region below. There is bone destruction and periosteal new bone formation. Some evidence of sequestration is present medially in the upper portion.

where the patient had complained of pain intermittently for one week previously. More complete roentgenographic study showed an extensive osteomyelitis in this site (Fig. 2). On the seventh postoperative day, marked right wrist drop was noted with involvement of the muscles of the right hand. This was believed to be either a "toxic" neuropathy or possibly

to be related to an early osteomyelitis in the right arm. A complete bone survey by roentgenogram at this time revealed further osteomyelitic foci in the left radius and both femurs (Fig. 3).

Much nausea and emesis developed from the Terramycin medication, and ten days postoperatively the patient was started on intravenous Aureomycin. This was supplemented by streptomycin two days later. Ulnar neuropathy on the left side became evident and was believed to be on the same basis as the right radial neuropathy noted earlier. Aureomycin was discontinued on the twenty-third postoperative day and parenteral Terramycin started which was continued for an additional twenty-one days. Repeat roentgenograms of the osteomyelitic areas showed healing, and mobilization of the patient was started. During the following week the patient became afebrile for the first time and remained so for an additional week at which time all antibiotic therapy was discontinued. Liver function studies at this time revealed direct serum bilirubin to be 0.16 mg. per cent and total bilirubin to be 0.90 mg. per cent with 1.3 per cent Bromsulphalein retention. Active physiotherapy was begun and the patient continued to improve rapidly, roentgenograms continuing to show healing of the osteomyelitis. The boy was discharged from the hospital on the 116th day.

Fig. 3.—Both femurs show a widespread, yet spotty, destruction of the cortex.

Outpatient follow-up was uneventful, the patient rapidly gaining weight and increasing activity to normal. Roentgenograms continued to show progressive healing of the bone lesions.

Eight months after operation the patient noted a small mass in the splenectomy wound which was not painful. The boy was afebrile. He was seen in the clinic at which time a 2.5 cm. by 2.5 cm. soft, nonreducible mass was palpated in the mid-portion of the wound. The skin over the mass was not erythematous or hot, but some tenderness was present in the area. Aspiration of the mass was nonproductive. It was felt that the mass should be observed for a short time. After three weeks of no change in the size or character of the

mass, re-examination revealed some erythema of the skin over the mass. Exploration was advised and the patient was readmitted to the hospital where under local anesthesia the area was opened. In the subcutaneous tissue layer, an abscess cavity was entered which contained approximately 5 ml. of purulent material. Culture of the material revealed *S. enteritidis*. The patient was discharged to home where intermittent hot soaks to the area were continued until clean healing was present. Postoperative follow-up has been unremarkable, the patient remaining in excellent health since.

COMMENT

A large number of individual case reports of Salmonella osteomyelitis have appeared in the literature in recent years, only a small fraction of which have been caused by *S. enteritidis*. The Salmonella infection in most of these cases has been ushered in by gastrointestinal symptomatology although such a clinical picture is not necessarily present. Over one-half of the cases of Seligman and associates[2] had gastroenteritis as the predominant clinical sign. Septicemia or typhoid fever-like symptoms, such as in the case presented here, were uncommon, occurring in only 13 per cent of the total series of Seligman. No contact or source of the infection could be found in the present case.

Smith[3] has pointed out the difficulty in differentiating radiographically the similar bone lesions of sickle-cell anemia and osteomyelitis. No localized bone lesions had ever been noted by roentgenogram in the present case prior to the final hospital admission. The sudden appearance, therefore, of areas of bone destruction with periosteal new bone formation and sequestration in the presence of septicemia made it obvious that osteomyelitis was present. Subsequent clearing of the lesions roentgenographically confirmed this impression.

Fortunately, the organism in the present case was sensitive to a large number of antibiotics so that when discontinuation of one was indicated because of adverse effects, another effective drug was substituted. The improvement and ultimate cure which resulted in the patient attests to the effectiveness of such a program.

The development of small splenic infarcts is not unusual in sickle-cell anemia, probably on the basis of thrombosis of vascular channels with hemolyzed blood cells. In the presence of septicemia such infarcted areas become perfect targets for abscess formation, as happened in the present case. Splenectomy here was of twofold benefit: drainage of the abscess; and perhaps alleviation of the hemolytic process since there are many advocates of splenectomy in patients with thalassemia or sickle-cell anemia. The absence of even minor hemolytic episodes since splenectomy indicates the benefit of the surgical procedure here.

During drainage of the splenic abscess there undoubtedly was much contamination of the wound with Salmonella organisms. Persistent viability of these organisms, however, for a period of eight months in the wound without symptoms is somewhat unusual. Fortunately the secondary wound abscess was well localized and did not act as a focus for the initiation of another episode of septicemia.

SUMMARY

A case of thalassemia with sickle-cell anemia in a patient who developed *Salmonella enteritidis* septicemia and osteomyelitis has been presented. Splenic abscess necessitated splenectomy, and a remote postoperative wound abscess containing viable *S. enteritidis* organisms followed which was drained without event.

REFERENCES

1. Giaccai, L., and Idriss, H.: Osteomyelitis Due to Salmonella Infection, J. Pediat. **41**: 73-78, 1952.
2. Seligman, E., Saphra, I., and Wassermann, M.: Salmonella Infections in Man—an Analysis of 1,000 Cases, Am. J. Hyg. **38**: 226-249, 1943.
3. Smith, W. S.: Sickle Cell Anemia and Salmonella Osteomyelitis, Ohio M. J. **49**: 692-695, 1953.

LATE HEMOPTYSIS FROM RIB SEQUESTRUM THIRTY-FOUR YEARS FOLLOWING EMPYEMA DRAINAGE

BENSON B. ROE, M.D., AND MORTIMER A. BENIOFF, M.D., SAN FRANCISCO, CALIF.

(From the Department of Surgery and the Department of Medicine, University of California School of Medicine)

THE differential diagnosis of hemoptysis in the absence of objective radiologic or bronchoscopic findings is a perplexing problem. Often the source of spontaneous bleeding is never established before it subsides. Occasionally, however, a clinician is faced with the necessity of emergency surgical intervention to control massive hemorrhage. When this situation arises, the surgeon's choice of which lung to explore may have to be made on the basis of a chance observation, if, indeed, he has any choice at all.

In most cases the cause of a large intrabronchial hemorrhage can be discovered or suspected from a careful history and physical examination and with plain chest roentgenograms. Confirmation of the character and location of the bleeding lesion usually requires either direct (bronchoscopic) or indirect (bronchographic) visualization of the bronchial tree. Nasopharyngeal or esophageal bleeding sources are sometimes deceiving and, of course, need to be considered. Occasionally, however, none of these studies yields significant information either because the presence of blood precludes satisfactory observation or because of a peculiar circumstance with seemingly satisfactory studies.

The following case is of interest because of the unusual character of the lesion, its long duration, and the repeatedly nonspecific x-ray and bronchoscopic findings.

CASE REPORT

History.—S. S., a 35-year-old white married man, was admitted to the hospital because of repeated bouts of massive hemoptysis. He described first experiencing a bubbling sensation in his chest and immediately thereafter coughing up variable amounts of grossly bloody fluid. This would eventually stop spontaneously and would be followed by bloody sputum for several hours.

The patient dated his illness to early childhood. At the age of 1 he had an episode of pneumonia which was followed by empyema requiring surgical drainage. Since that time and at varying intervals up to the age of 18, he had coughed up a mouthful of bloody sputum without associated pain or other symptoms. These episodes were followed by a symptom-free interval of about seventeen years when he again had a bout of significant hemoptysis eight months before consulting us. He was bronchoscoped at that time and a small area in his bronchus was cauterized. Two other episodes of major hemoptysis occurred, one one week and the other one day prior to being seen.

Received for publication April 19, 1955.

The patient reported having a chronic cough for many years, up until about the age of 18 when he stopped smoking and these symptoms improved significantly. Repeated tests for tuberculosis in his youth and childhood had been completely negative.

His general health had been excellent. His past history was essentially noncontributory except for the knowledge of having a heart murmur for many years. He had, however, never been physically handicapped in relation to his work or athletics. He had no difficulty in breathing, no orthopnea or ankle edema. He was born and reared in the San Francisco Bay area and had no history of residence in an area endemic for coccidioidomycosis.

Fig. 1.—Plain chest film demonstrating left upper lobe density with a ringlike shadow. Streaky parenchymatous shadows not present on previous films presumed to be product of hemorrhage.

On physical examination the patient was noted to be well developed and healthy. Significant findings were confined to the chest. There was a well-healed scar over the left inferior posterolateral chest from the previous thoracostomy. Respiratory movements of the left hemithorax were slightly restricted, and the right chest was somewhat larger than the left. The lung fields were resonant to percussion, and no abnormal breath sounds could be found. A loud to-and-from murmur was heard over the entire precordium, loudest over the left third intercostal space. Blood pressure was 120/80, and the pulse was 80 with regular rhythm.

Chest roentgenograms revealed obliteration of the left costophrenic angle and pleural thickening over the left lower chest. There was bony bridging

between the eighth and ninth ribs posterolaterally at the site of the old thoracostomy. There was a poorly defined, incomplete, ringlike density noted in the left upper lung field. The heart was normal in size and shape.

The patient was bronchoscoped and all segmental divisions of the bronchial tree were satisfactorily visualized and found to be entirely normal. No evidence of bleeding was noted. Adequate Lipiodol bronchograms were carried out and were not remarkable except for one spot film which showed an area of incomplete filling in the left upper lobe with an adjacent area of cylindrical bronchiectasis. A calcified aortic valve was demonstrated fluoroscopically.

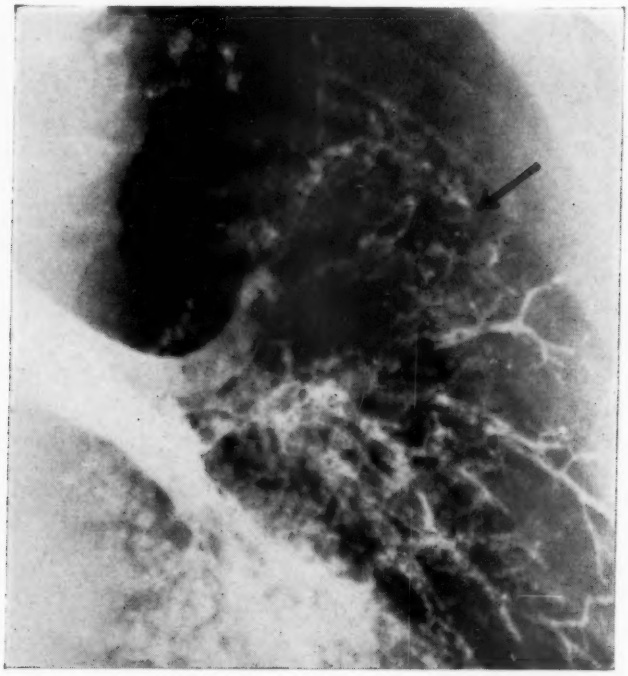


Fig. 2.—Spot film of left bronchogram showing minimal bronchiectasis in upper lobe segment and incomplete filling.

While under hospital observation, he had another episode of significant hemoptysis when one of us was fortunate enough to observe and examine him. He localized the gurgling to the left upper chest posteriorly where definite gurglings were audible with a stethoscope.

The extent and persistence of the bleeding were sufficient to demand surgical intervention despite the lack of a definite diagnosis. Bronchiectasis or vascular anomaly was considered the most likely possibility. Thoracotomy was performed on Oct. 7, 1952. The pleural space was found to be free of adhesions except in the region of the previous rib resection where the lung was freed up without difficulty. In the apical posterior segment of the left upper lobe, a firm oval density, measuring 1 by 1 by 2 by 2 cm. could be palpated. The remainder of the lung was normal to appearance and palpation. An apical posterior segmental resection was carried out, the lesion was examined by the pathologist, and the chest was closed.

Pathology Report.—

Gross examination: Specimen submitted consists of a portion of lung weighing 38 grams and measuring over all 11 by 8 by 3.5 cm. Pleural surface is clean and slightly streaked with anthracosis. The major branches of the bronchial tree taper in a normal manner with a smooth intact mucous membrane, except one of the two principal larger divisions which shows in proximal portion a saccular dilatation measuring approximately 8 mm. in diameter. Occupying this thick-walled dilatation were two mineral masses measuring, respectively, 11 by 5 and 15 by 6 mm. with an average thickness of 1 to 2 mm. They have the gross character of bone. The surrounding bronchial wall is moderately thickened and firm. Section reveals no evidence of malignant change. The remainder of the pulmonary parenchyma is normal.

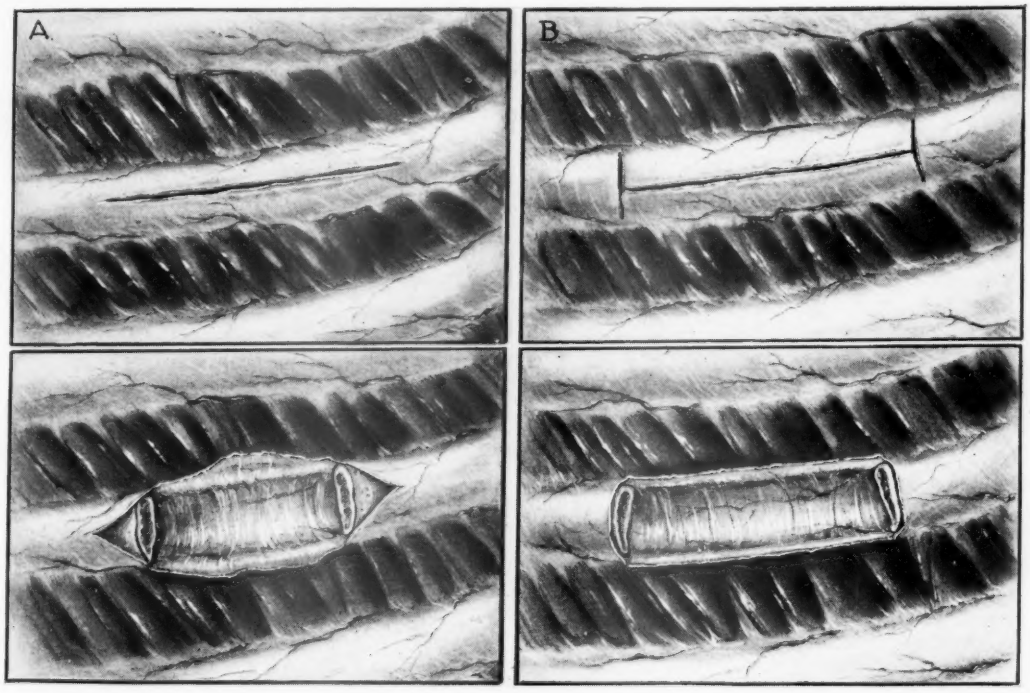


Fig. 3.—(*A*), Simple periosteal incision resulting in denuded triangles of bone following resection, (*B*), flapped incision showing bone ends remaining covered with periosteum following resection.

Microscopic examination: The dilated bronchus is lined in part by respiratory epithelium and in part by stratified squamous epithelium. There are a number of areas of denudation present with a richly vascular base. The surrounding wall is densely infiltrated by lymphocytes to a depth of 3 or 4 mm. with marked proliferation of fibrous tissue.

Diagnosis.—Foreign bodies, bronchus: (a) bronchiectasis; (b) bronchial ulceration; (c) squamous metaplasia, bronchus; (d) periobronchial fibrosis.

It is of interest to speculate on the origin of the decalcified bony foreign body. Aspiration is fairly well ruled out on the basis of a reliable negative history from both patient and family, and the size of fragment which would lodge in a major bronchus even of an adult.

Failure of this material to be visualized roentgenologically is curious. The history of rib resection for empyema shortly before the onset of hemoptysis is of probable significance. Sequestration of exposed deperiostealized rib is known to occur particularly in the presence of sepsis. It seems more than likely that this patient had a fragment of necrotic rib sloughed intrathoracically following his empyemia drainage, with subsequent incorporation in the pulmonary parenchyma. Churchill[1] has emphasized the importance of incising rib periosteum in a manner which prevents denuding bone beyond the point of resection (Fig. 1). This case serves admirably to illustrate his point.

SUMMARY

Rib resection in the presence of sepsis can result in sequestration of transected costal ends. When these fragments lodge in the lung parenchyma, a foreign-body reaction results and may be the cause of hemoptysis. A case in point is presented with an unusually long history of hemoptysis. Proper technique of rib resection to avoid this complication is illustrated.

REFERENCE

1. Churchill, E. D.: The Technic of Rib Resection and Osteomyelitis of the Rib Ends, J. A. M. A. 92: 644 (Feb.) 1929.

Editorial

Is There Something Wrong With Residency Training Programs?

A SURGEON recently sent me a copy of a letter from one of his former residents. It is a thought-provoking communication. Here it is:

Dear _____:

I thought that you and the resident staff might like to hear about my impressions and experiences after the first couple of months on the job in a small surgical service—it certainly is an awakening, and one really learns in a hurry what his shortcomings are. For one thing, both administratively and professionally, there is a big void here as compared to the ease with which we could formerly present our problems to a more authoritative, experienced, and responsible individual, and I find myself thankful now for having been allowed such a free latitude in making decisions, with relatively few corrections, revisions or reversals. The sense of responsibility and self-confidence so gained has certainly helped me to adapt to the new situation.

Then, too, the term "General Surgeon" has come into focus at last, and the loopholes in training and my own guilt in sloughing of the subspeciality rotations is coming home to roost. Here we're not endowed with great numbers of super-refined specialists, and everything that can be construed as surgical comes under my service with poor old me accepting the final responsibility. Thus the loopholes—of course, I'm not expected to be able to refract eyes, or wash out sinuses or such, but I find that EENT, of which I had none, makes up a tremendous amount of the outpatient load. I fortunately have a very capable physician to handle this outpatient work, but still I have to send children a couple hundred miles for simple tonsillectomies. Recently, I reduced my first nasal fracture. Here are two procedures that anyone should be able to do, but I hadn't taken the time to observe them in four years at _____. So I plan on trying to get a couple of weeks TDY during the busy season next fall to observe ENT, so that I don't feel so nervous and dependent when one of these simple problems comes up. I certainly would encourage the residents to renew their acquaintance with ENT before leaving _____.

It may come as a profound shock to some of the residents to learn that I was the obstetrician and gynecologist here for about 3 weeks, and still take the emergency Ob-Gyn call a third of the nights. This came about because of a change of Ob-Gyn men which was not perfectly coordinated, and my call is now effective because it's just not right to have one man on call twenty-four hours a day, seven days a week, with no time to call his own. I know anyone can "catch" a baby, but it's helpful to keep up with the niceties of delivering them. And as fate would have it, we had two emergency sections last month, one for previa with hemorrhage, and one for disproportion. A few nights or a couple of weeks in the labor room and delivery room upstairs might be of real value for some of those potentially in a spot similar to mine. Happily for me, I had previously done Ob-Gyn at _____.

G.U. contributes its share of cases, particularly chronic prostatitis, cystitis, strictures, stones, and such. And we've found that it's nice to have used that cystoscope freely on the G.U. Rotation.

Another thing I sorely miss is not having had recent responsibility for severe burns —by that I mean it just doesn't suffice me to read or listen to the management, or go in and watch _____ débride and graft and dress burns. Fortunately we've had no bad burns as yet, but obviously it's only a matter of time.

Everything is important—we have no pathologist here so I do tentative readings on all surgical specimens, or else we'd have no diagnosis for a month or more. Plastic repairs and head injuries are ours.

But the thing that I've repeatedly blasted myself for being so stupid about is so-called orthopedics. The knees and backs and strains and sprains don't bother me, but I've certainly been hypnotized for years into thinking that fractures were orthopedic problems. I've almost done an entire about-face, and now believe that if fractures are not exclusively in the province of the general surgeon, they are certainly equally general surgical problems. My five months of orthopedics were spent on the osteo ward in the midst of the Korean affair, and I don't imagine I saw ten closed reductions the whole time there. And I sat like a fool while at least fifty or more fractures that I saw were being set in another room or building without making the effort to see or do the reduction! How stupid can one be—20 to 25% of our load of major cases are fractures and we don't have a highly paid, muscular orthopedist. If I had it to do over, I would insist on seeing and either assisting or reducing every fracture that came in when I was on surgical call, and I know that I'd made every effort to spend my orthopedic time on the fracture and amputation section. It would be best if every other fracture were admitted to General Surgery, particularly if the other residents have been as blind as I.

Well, that's the most of it—I sit here now full of regrets at not having taken full advantage of the tremendous wealth of material that flows through _____. I know that it's impossible to arrange a rotation whereby all the residents can become skilled in all these fields—it's entirely up to them to put in the extra time and effort at the cost of a nagging wife, a missed party or show, or less sleep; stealing, scrounging, looking on or doing while some other soul is goofing or sleeping. If they don't do it that way they're going to end up like myself—getting by with nerve and book-larnin', until he catches up with the lost opportunities.

You'll notice, of course, that I haven't said a word about any feeling of inadequacy about chest or abdominal surgery, or the other procedures we include in the narrower meaning of General Surgery. I'm just as sure of myself and cocky about that as ever, and could still approach a gastric resection for bleeding or a lobectomy for TB. with less misgivings than I have with a relatively simple trimalleolar fracture, because I'm sure of my training and ability to handle that. I'm extremely grateful, too, for the single short month on Anesthesiology that has more than once helped me to bail my anesthetist out of difficulty. The one thing that has come home to me is that I didn't "finish" training—I just graduated into a rougher, tougher school where myself, experience, and hard knocks are going to be the teachers. . . .

Those responsible for residency training in general surgery might well begin to ask themselves whether a well-rounded program of training is not, after all, what we ought to aim at. When a resident is trained so that he can do with ease abdominal and thoracic surgery, is he, in reality, a general surgeon, or is he, like so many of the men being trained, simply an abdominal and thoracic surgeon, or even, one or the other?

Surely every resident ought to know something about urologic surgery and neurosurgery, and, in addition to these, something about gynecologic surgery and the surgery of fractures and trauma in general. Are we not stretching a point when we call the men who have failed to have such really broad training general surgeons? Many of us have repeatedly stated that the surgical specialist should have an adequate background in general surgery. The surgical specialist boards are recognizing the necessity of such training. Is it not also true that the young general surgeon will be more adequately trained if he has at least had background training in the important surgical specialties?

—I. S. Ravdin

Recent Advances in Surgery

CONDUCTED BY ALFRED BLALOCK, M.D.

THE SYNDROME OF THE CHRONIC LEG ULCER

THE PHLEBODYNAMICS OF THE LOWER EXTREMITY; PHYSIOLOGY OF THE VENOUS VALVES

STANLEY C. FELL, M.D.,* HENRY D. MCINTOSH, M.D.,** AUBREY T. HORNSBY, M.D., CHARLES E. HORTON, M.D., JAMES V. WARREN, M.D., AND KENNETH PICKRELL, M.D., DURHAM, N. C.

(From the Departments of Surgery, Medicine, and Radiology, Veterans Administration Hospital, and Duke University School of Medicine)

DESPITE extensive investigation the pathogenesis of the chronic leg ulcer remains the subject of considerable debate. This dilemma results in part from the incomplete understanding of the phlebodynamics in the normal lower extremity.

Most chronic leg ulcers are considered to be secondary to deep vein thrombosis.[1] A small group of patients, however, develops ulcers without a history of previous thrombophlebitis, but in association with varicose veins, obesity, severe leg trauma, arteriosclerosis obliterans, cellulitis, lymphangitis, peripheral neuropathy, sickle-cell anemia, or arteriovenous fistula.[2] How prior thrombosis or these associated conditions alter the function of valves, communicating veins, venomotor tone, supporting structures, and/or other unknown factors and lead to the development of a chronic leg ulcer is at present uncertain.

In the present study venographic observations were correlated with hemodynamic measurements in an attempt to define alterations of normal phlebodynamics associated with chronic ulceration of the lower extremity. Particular attention was directed to the elucidation of valvular function in the normal and pathologic extremity.

MATERIALS AND METHODS

This study is based on observations of 39 subjects. Both legs were studied in some subjects; in others, only one extremity was studied. Therefore, a classification of material based on extremities rather than individuals is necessary.

Thirty-three extremities were normal by history and physical examination. These comprise the normal group. Twenty-three extremities comprise the lower leg syndrome (chronic leg ulcer) group. These are subdivided as to history in Table I. An additional three extremities had primary varicose veins without leg ulcer.

A percutaneous technique was used for venography and venous pressure measurements. Femoral venipuncture was performed with a thin-sheath 16

This investigation was supported in part by Research Grant H-1217(C3) from the National Heart Institute of the National Institutes of Health, United States Public Health Service and the American Heart Association.

Received for publication May 4, 1955.

*Present address: Montefiore Hospital, New York, N. Y.

**This study was carried out during Dr. McIntosh's tenure of an American Heart Association Fellowship.

gauge cannula and inner needle.* Approximately 4 cm. of polyethylene tubing† was introduced through the cannula into the vein. The cannula was then withdrawn. It was occasionally necessary to expose surgically the saphenous vein at the ankle for introduction of the polyethylene tubing. The popliteal vein was catheterized by the same percutaneous technique, using the method of Veal and Hussey[3] for localization of the vein.

TABLE I. CLINICAL CLASSIFICATION OF EXTREMITIES

I. Normal controls		33
II. Lower leg syndrome (chronic leg ulcer)		
1. Prior thrombophlebitis	13	
2. Prior leg trauma (no history of thrombophlebitis)	6	
3. "Primary" varicose veins	2	
4. Neurological disease	2	
Total		23
III. "Primary" varicose veins (no leg ulcer)		3

Retrograde femoral venography was performed in 57 of the 59 extremities; antegrade venograms, via the saphenous vein, were performed in 19 extremities. Popliteal venograms were made on five occasions. All venograms were made with the supine subject tilted 65 degrees, head upward.

Retrograde venograms were performed by slowly injecting 25 ml. of 30 per cent Urokon through the femoral catheter under fluoroscopic observation. While the patient performed quadriceps contractions for ankle exercises, the rapidity and degree of clearing of the opaque material were assessed. Following fluoroscopy, an additional 40 ml. of Urokon was injected slowly, while three overlapping stereoroentgenograms were made of the entire extremity.

When supplemental information was desired, especially concerning communicating veins, stereoscopic antegrade radiograms were made. With this technique, a tourniquet was placed about the lower leg 10 cm. proximal to the tip of the catheter lying in the saphenous vein, for the purpose of forcing the opaque material into the deep venous system. Popliteal venography was utilized in a small group of subjects. A tourniquet was not used in this procedure.

Simultaneous femoral and saphenous venous pressure measurements were recorded from 18 extremities; 11 were clinically normal, and seven exhibited signs of the lower leg syndrome. From four of the normal extremities and one postphlebitic extremity, simultaneous femoral, popliteal, and saphenous venous pressure measurements were recorded. No pressure measurements were made within one hour following venography.

Pressures were recorded during cough, the Valsalva maneuver, quadriceps contraction, and marking time by suitable Statham strain gauges on a four-channel direct-writing Sanborn polyoscillograph. The gauges were secured to the extremity so that the vein was not compressed, with the transducer membrane at the level of the catheter tip.

*T479 LNR Needle, Becton, Dickinson & Co., Rutherford, N. J.

†Polyethylene Medical Tubing PE 90; internal diameter 0.86 mm., outside diameter 1.27 mm.; Clay Adams Co., New York, New York.

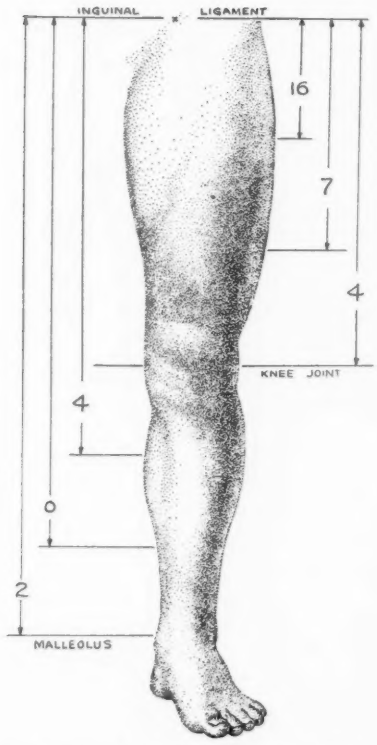


Fig. 1.—The numbers refer to the frequency of respective levels of retrograde opacification of deep veins encountered in 33 normal extremities. The subdivisions are based on thirds of the thigh and leg.

Fig. 2.—R. C., a 24-year-old normal subject. Retrograde venogram via a catheter in the common femoral vein demonstrates abrupt termination of reflux at valves in the upper third of the superficial femoral vein.

RESULTS AND COMMENTS ON VENOGRAPHY

I. *Normal Group: 33 Extremities.*—In 27 of 33 clinically normal extremities, the contrast material refluxed from the femoral injection site to varying

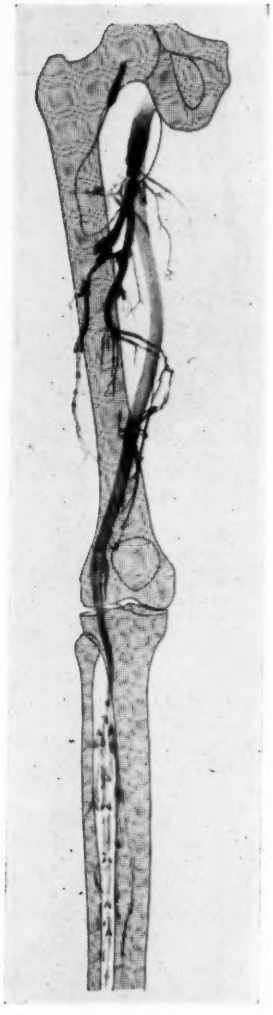


Fig. 3.

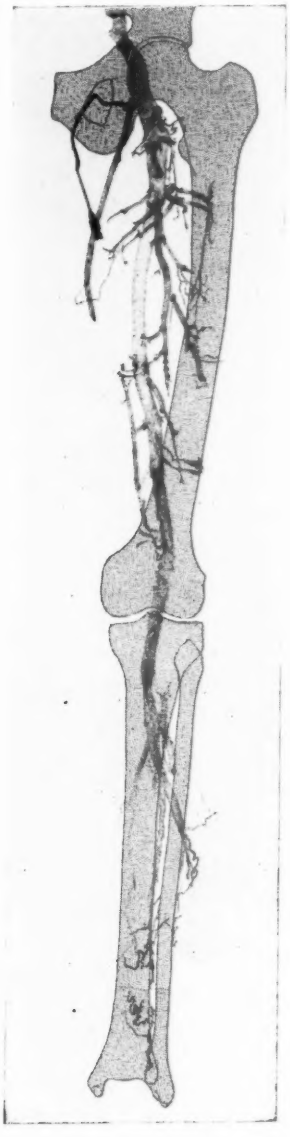


Fig. 4.

Fig. 3.—J. E., a 26-year-old normal subject. Observe the retrograde opacification of the deep veins, to the level of the ankle joint, terminating in the communicating veins. The superficial system is not visualized. The artist's tracing of the roentgenograms demonstrates the density of the dye in the venous system.

Fig. 4.—J. W., a 61-year-old man with bilateral chronic leg ulcers; retrograde left femoral venogram via the dorsal vein of the penis.[29] There is no evidence of prior thrombophlebitis. Numerous valves are by-passed during reflux to the ankle. The superficial system fills via tortuous dilated communicating veins. Deep thrombophlebitis occurred in this extremity 7 days after contralateral vein stripping and 9 days after venography. (See Figs. 5 and 6.)

levels of the superficial femoral vein above the knee joint. The remaining six clinically normal extremities demonstrated reflux below the level of the knee joint (Fig. 1).

Regardless of the degree of reflux, there was no apparent filling of the superficial system from the deep veins (Figs. 2 and 3). It was not uncommon, however, to visualize faintly opacified communicating veins. These veins had small diameters and were not tortuous. At least two, and occasionally as many as eight, valves were visualized in the superficial femoral vein.

The lowermost point of reflux of contrast material could be determined within a few millimeters in 27 of the 33 normal extremities. In 18, reflux was apparently halted by a valve station, while in 9 the arrest of contrast medium was noted in a segment of vein below a valve.

Fig. 5.—Retrograde femoral venogram of same extremity as in Fig. 4, 3 months after deep thrombophlebitis. There is irregularity and narrowing of the common femoral vein and thrombotic occlusion of the superficial femoral vein. Note that reflux is halted at the site of thrombosis.

II. *Lower Leg Syndrome: 23 Extremities.*—

1. *Prior thrombophlebitis:* Radiographic evidence of deep venous thrombosis by retrograde and/or antegrade study was demonstrated in 12 of the 13 extremities in which a history of prior thrombophlebitis had been obtained (Figs. 4 and 5). Deep vein block was not demonstrated in the remaining extremity, perhaps due to inadequate study. Conversely, a femoral vein thrombosis was demonstrated in one of six extremities with an ulcer and a history of antecedent trauma but no history of thrombophlebitis (Fig. 6). The correlation of venographic findings, history, and physical findings is therefore quite good in this small series.

The site of thrombosis in these 13 extremities could be demonstrated by the retrograde technique alone in 11 extremities. In no extremity was the thrombosis observed only below the level of the popliteal vein. The reflux of contrast material was arrested at the area of deep vein thrombotic occlusion in eight extremities; in two, retrograde flow occurred through capacious collaterals by-passing the block; in the remaining case, retrograde filling of

Fig. 6.—Retrograde right femoral venogram of same patient as in Fig. 4. There was no history of prior thrombophlebitis in this extremity. The venogram demonstrates thrombosis and partial recanalization of the superficial femoral vein.

the leg veins occurred through a recanalized segment of the superficial femoral vein. Therefore, superficial femoral vein ligation as a therapeutic measure to prevent deep venous reflux could have no utility in 10 of these 11 extremities.

2. *Prior leg trauma:* As noted previously, a thrombosis of the femoral vein was demonstrated in one of the six extremities with an associated history suggesting nonphlebitic posttraumatic ulceration of the lower leg. Relatively uniform retrograde venographic findings were present in the remaining five extremities. All demonstrated massive reflux of contrast medium down the femoral system into the deep leg veins (Figs. 7*A* and 7*B*). The caliber of the femoral vein and the number of valves visualized did not differ significantly from the accepted normal. There was no evidence suggestive of prior thrombosis and "smooth recanalization." Passage of opaque material through tortuous communicating veins into varicose superficial leg veins was observed in all but one extremity (Patient R. W.).

3. *"Primary" varicose veins:* In two patients with the lower leg syndrome associated with severe "primary" varicose veins, the contrast material refluxed to the upper third of the superficial femoral vein in one, and to the middle third in the other. Antegrade venograms demonstrated normal deep veins in both cases, although dilated communicating veins were noted in one extremity.

4. *Neurological disease:* Two patients with unilateral chronic leg ulcers associated with peripheral neuropathy, when studied by the retrograde method, demonstrated arrest of the contrast material at valve stations in the upper third of the superficial femoral vein. Despite extensive ulceration of the lower leg, these studies could not be distinguished from venograms of clinically normal extremities.

III. *"Primary Varicose Veins (No Leg Ulcers): Three Extremities.*— Retrograde venography demonstrated reflux down the superficial femoral vein to the lower third of the thigh, the upper third of the leg, and the lower third of the leg, respectively, in these three extremities. Dilated tortuous communicating veins were noted radiographically in two of these three extremities.

Communicating veins: The condition of the communicating veins may be evaluated by either the retrograde or the antegrade technique. In the latter procedure, a tourniquet is routinely applied to the lower leg proximal to the site of injection of the saphenous vein at the ankle, thus forcing the opaque medium into the deep venous system. If the medium returns to the superficial system proximal to the tourniquet, incompetent communicating veins may be suspected.[2]

It is generally assumed that the normal communicating veins convey blood chiefly in one direction, that is, from the superficial to the deep system. Following injury to these veins, the flow may occur in both directions and the veins are considered incompetent.

In this study it was not uncommon, however, to demonstrate by both the retrograde and the antegrade techniques partial filling of the communicating veins in the normal, relaxed extremity. These veins appeared quite narrow and straight. The superficial system was not opacified by such techniques. It is possible that opaque material did reach the superficial system but the concentration was so dilute that it was not radiographically visible.

In contradistinction, markedly dilated and tortuous communicating veins were demonstrated in 16 of the 23 extremities with the lower leg syndrome. These veins were considered to be functionally incompetent. The superficial system was visualized in such cases by both the antegrade and the retrograde

Fig. 7A.—C. L., a 55-year-old man with lower leg syndrome but no history of prior deep vein thrombosis. Retrograde femoral venography was performed, before and after circumferential excision of the full thickness of the ulcerated skin and fascia of the lower one-half of the leg and split-thickness grafting. Both studies demonstrated massive reflux of contrast material into the deep leg veins. The preoperative venogram of the leg (fourth panel) illustrates the markedly dilated communicating and superficial veins which filled via the deep system. The postoperative venogram of the leg (third panel) demonstrates that the pathologic veins in the superficial compartment have been removed.

techniques. Such visualization was due to the reflux of sufficient opaque material in a concentration adequate to produce radiographic opacification. An adequate concentration of opaque material in the superficial system is apparently possible only when the communicating veins are dilated.

The opacified deep venous system was observed fluoroscopically during muscular contraction. In normal subjects, the opaque medium was not observed to pass from the deep to the superficial system. However, in subjects with the

lower leg syndrome, the opaque material routinely passed through dilated communicating veins into the superficial system. The venous system did not appear to become clear of the opaque material as rapidly from extremities involved with the lower leg syndrome as normal extremities.

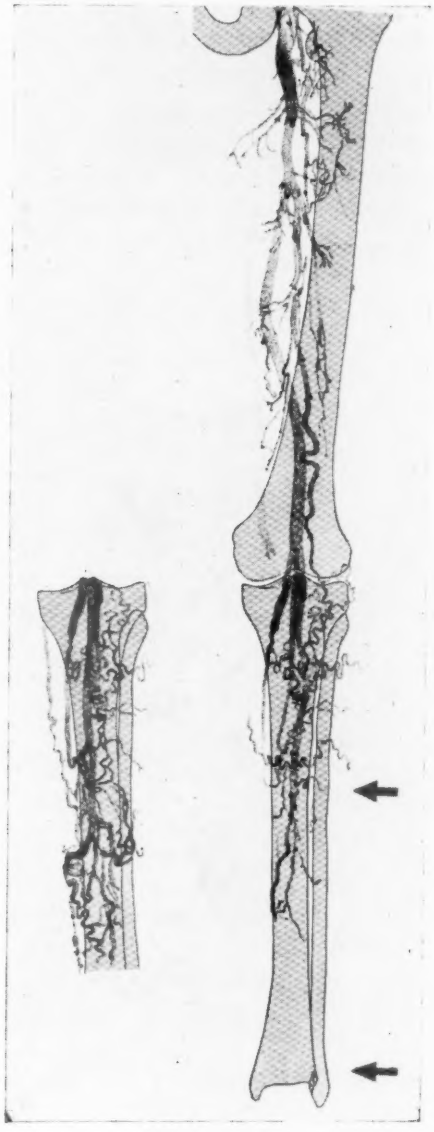


Fig. 7*B*.—Same subject as in Fig. 7*A*. The inset on the left is a drawing of the opacification of the leg veins on the preoperative retrograde femoral venogram. On the right is the postoperative venogram. Arrows indicate the extent of the skin and fascial excision.

Venographic technique and interpretation: Venography by the described techniques may be accomplished through the femoral and saphenous veins in almost every patient. The popliteal vein technique is more difficult and contributed little information not obtained by the antegrade method.

The retrograde and antegrade flow of opaque medium, as noted previously, may be easily observed fluoroscopically. It is a useful procedure in determining the extent of retrograde flow of contrast material, and the rapidity and degree of clearing with exercise. Information obtained by fluoroscopy is, however, chiefly supplemental to that obtained venographically.

A fallacy in venographic interpretation is the assumption that failure of a particular vessel lumen to be opacified is due to thrombotic obliteration. Contrariwise, good filling of major veins does not indicate all significant venous channels are filled. It is frequently necessary to use both antegrade and retrograde techniques to evaluate an extremity adequately. Stereoroentgenography is a valuable aid in identification of veins. Without such stereofilms it is often impossible to determine whether a vein or group of veins belongs to the deep or superficial system.

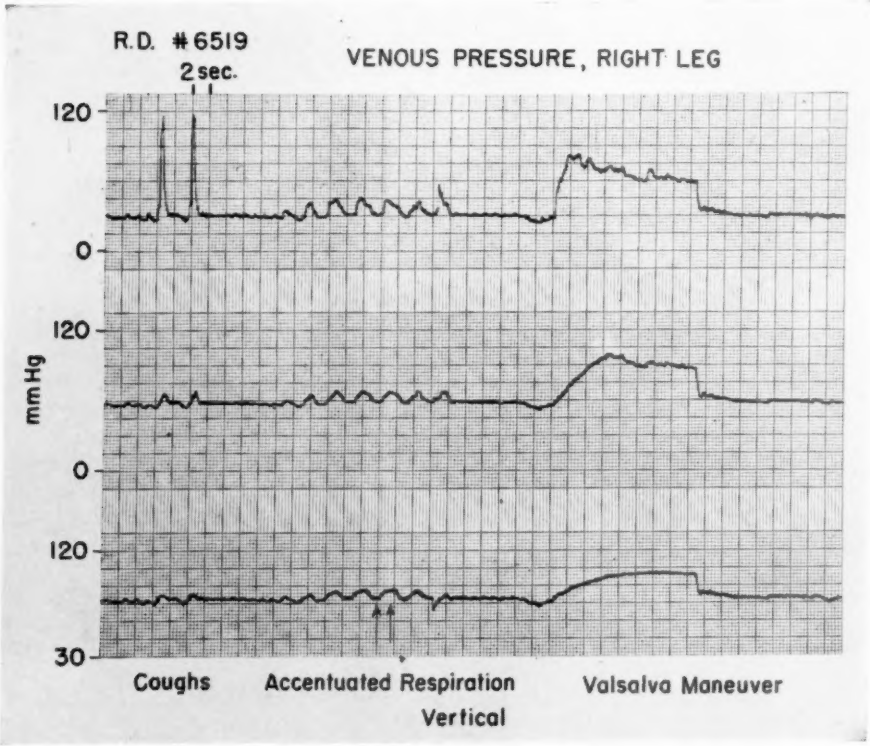


Fig. 8.—Recorded from above downward are the superficial femoral, popliteal, and saphenous venous pressures in a subject with clinically and venographically normal extremities. In all illustrations the pressures were recorded at the level of the catheter tip and variations are changes from hydrostatic pressure. The rhythmical fluctuation in the hydrostatic pressure recorded at the three sites was due to an arterial impulse propagated through the venous system while the valves were open. The inspiratory phase of respiration is marked by arrows. (See text.)

RESULTS AND COMMENTS ON VENOUS PRESSURE MEASUREMENTS

During motionless standing, the venous pressure in the lower extremity equals the hydrostatic pressure of the column of blood to the right auricle. Such a correlation is present in the normal[4-10] as well as the pathologic extremity.[7-12] An exception to this observation is said to occur during the acute

phase of thrombophlebitis; the venous pressure below the obstruction is greater than the hydrostatic pressure until reflux spasm subsides and adequate collaterals develop.[13]

During motionless standing, "a," "c," and "v" waves were identified in the saphenous tracing (with proper standardization of the recording system). Similarly, a pulse wave, apparently produced in the venous column by an adjacent artery, was observed in the femoral, popliteal, and saphenous venous pressure tracings of several subjects (Figs. 8 and 9). Changes of intrathoracic pressure up to 10 mm. Hg, produced by accentuated respiration, were transmitted equally well to the deep and superficial system.

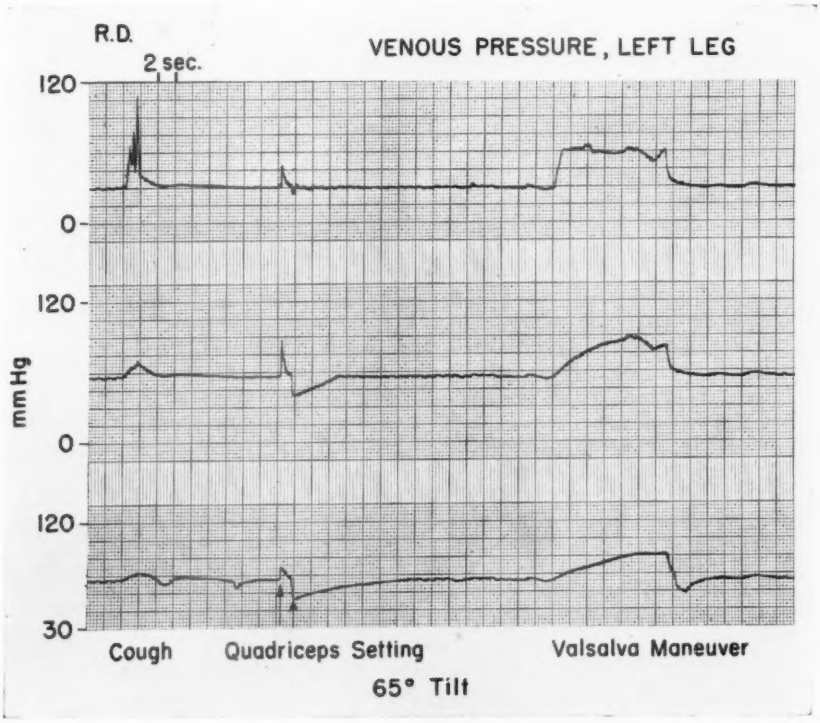


Fig. 9.—The same patient as in Fig. 8, contralateral normal extremity. The first arrow in the saphenous tracing indicates the onset of quadriceps contraction; the second arrow indicates the completion of relaxation. Again note the arterial pulse waves visible in the three tracings when the pressure was hydrostatic. (See text.)

These observations suggest that during motionless standing the continuity of the column of blood from the foot to the right auricle is functionally uninterrupted by valves. The valves in normal subjects are maintained open by the heartward flow of blood. In the pathologic extremity the functional competency of some valves may have been destroyed; those remaining are maintained open as in the normal subject. However, if in normal subjects respiration was more forced or if the intrathoracic and intra-abdominal pressure was increased by coughing or by the Valsalva maneuver, transmission of the pressure was promptly observed in the femoral tracing but not in the popliteal or saphenous. Subjects with the lower leg syndrome responded differently.

Cough.—In normal subjects single coughs of 0.3 to 0.6 second duration and an intensity of 60 to 80 mm. Hg, as recorded from the femoral vein, were transmitted poorly, if at all, to the popliteal and saphenous veins (Figs. 8 and 9). This was true regardless of the degree of radiographic reflux previously observed in the deep system.

The pressure change produced by cough in subjects with the lower leg syndrome was propagated to the popliteal and saphenous veins in almost undiminished intensity and without significant delay (Fig. 10). One exception to this observation was Patient R. W., in whom there was no venographic evidence of incompetent communicating veins.

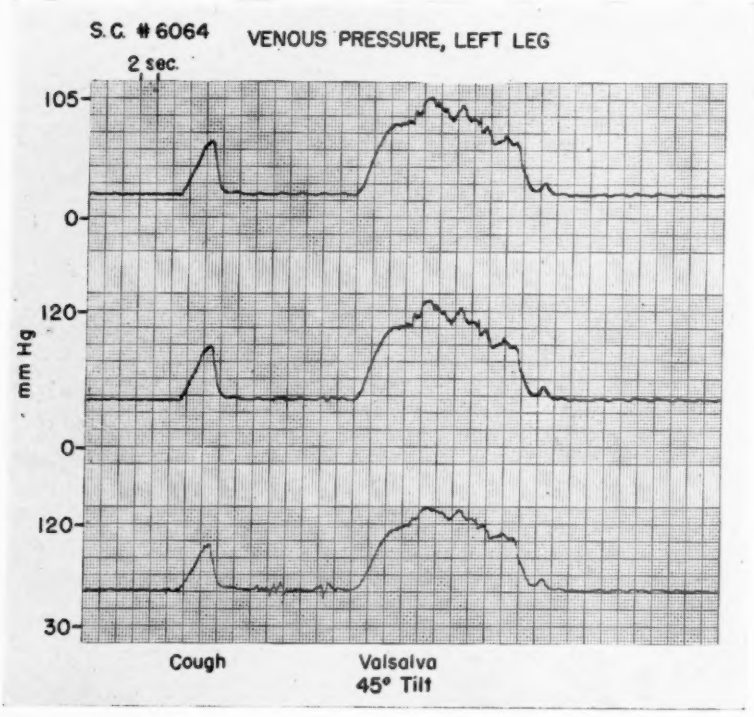


Fig. 10.—A postphlebitic extremity with a partially recanalized superficial femoral vein on venography. Note that the effect of increased intra-abdominal pressure recorded in the femoral vein is transmitted essentially undamped and without lag to the popliteal and saphenous veins. Application of tourniquets did not alter the pressure transmission.

The cough apparently "squeezed" blood peripherally from the abdomen and the sudden reversal of the gradient of flow closed competent valves. Therefore, the change of intra-abdominal pressure was not transmitted distally to the popliteal or saphenous veins. The absence of such competent valves permitted distal transmission. Whether a single valve or a number of valves is necessary to prevent transmission is at present uncertain.

Valsalva Maneuver.—When the Valsalva maneuver was performed, the femoral venous pressure in all subjects rose abruptly 40 to 80 mm. Hg above the hydrostatic pressure and usually remained at that level for the duration of

the strain. In some subjects there was, however, a slight delay (one to two seconds) in reaching the maximum pressure rise. It will be seen that such a lag probably is due to the catheter tip being distal to a competent valve in the superficial femoral vein.

The popliteal pressure was recorded simultaneously with the femoral pressure in four normal subjects. It rose gradually over a period of three to twelve seconds until the increment of pressure rise above the hydrostatic level equalled the rise of femoral pressure (Fig. 8). If the strain was continued, the popliteal and femoral pressures paralleled one another. If during the Valsalva maneuver the intensity of the strain was again suddenly increased above about 10 mm. Hg, the femoral pressure rose suddenly to the new level. The popliteal pressure rose gradually until an equal increment of pressure rise was attained. The femoral and popliteal pressures then paralleled each other for the duration of the strain at the newly attained pressure level. They changed simultaneously without lag during minimal fluctuations of intra-abdominal pressure (less than 10 mm. Hg). The femoral and popliteal venous pressures were parallel during a Valsalva maneuver only when the increment of pressure rise at both sites was equal.

The popliteal pressure was recorded during the Valsalva maneuver in one subject with the lower leg syndrome whose venograms demonstrated a thrombosed but partially recanalized superficial femoral vein. In this extremity, there was no lag in the rise of the popliteal pressure; it constantly paralleled changes of the femoral pressure (Fig. 10).

These observations suggest that during the initial phase of the Valsalva maneuver, as during a cough, blood is "squeezed" peripherally and valves, if present, are shut. Once the pressure rise in the popliteal vein equalled the pressure rise in the femoral vein, the gradient of flow became heartward and the valves opened. In the pathologic extremity, the valves are incompetent. Blood can, therefore, almost instantaneously be "squeezed" distally.

The delay during the Valsalva maneuver in rise of the popliteal pressure and thus the opening of the valves, varied considerably in the same individual with successive observations. This delay probably reflects the time required for arterial inflow[14, 15] and increased venomotor tone to afford the increment of pressure rise necessary to exceed the increased intra-abdominal pressure and thereby to re-establish the centripetal flow of blood.

With the onset of the Valsalva maneuver, the saphenous pressure in 11 normal subjects rose gradually in an essentially straight-line manner (Figs. 8 and 9). As the strain was maintained, the saphenous pressure occasionally stabilized at a new level; however, it never (under the conditions of this study) rose as high as the popliteal or femoral pressures. Minor fluctuations of abdominal pressure during the sustained strain which were observed in the femoral and popliteal tracings were not observed in the saphenous tracing.

Five of six extremities with the lower leg syndrome in which simultaneous femoral and saphenous pressures were recorded exhibited essentially parallel

rises of pressure at the two sites during the Valsalva maneuver. Application of tourniquets at various levels of the thigh and leg did not alter the pressure transmission to the saphenous vein.

The sixth extremity in the lower leg syndrome group (R. W.) responded to the Valsalva maneuver as well as to cough in the manner described for normal subjects. Radiographically, there was no evidence of incompetent communicating veins in this subject. This patient, however, had extensive ulceration of the lower leg.

These observations suggest that when communicating veins become functionally incompetent, changes of intra-abdominal pressure are freely transmitted to the ankle. However, such changes of abdominal pressure are not transmitted through competent valves. It is possible that alterations of the venomotor tone of communicating veins, produced indirectly by straining, reinforce the valves of the communicating veins in preventing retrograde pressure transmission.

These simultaneous measurements of the femoral (which served as a base line), popliteal, and saphenous venous pressures show that normal closed valves effectively prevent the distal transmission of changes of intra-abdominal pressure. The valves of the deep venous system may be opened, however, while the intra-abdominal pressure is elevated, provided the pressure below the valve exceeds the elevation of intra-abdominal pressure. Heartward flow of blood would then be resumed.

These observations are at variance with those of Hojensgard and Sturup.[11, 12] They suggested that retrograde pressure transmission was not prevented by normal valves. These observers did not record a base line (as in this study, the femoral pressure) with which to compare changes in the recorded pressure. They considered any rise of venous pressure as representing a transmission of pressure from the abdomen.

Quadriceps Contraction.—A single quadriceps contraction in the upright subject, maintained for two to three seconds, afforded an excellent opportunity to evaluate the phlebodynamics of the normal lower extremity (Fig. 9). With muscular contraction, the pressure rose rapidly in the deep and superficial system. As the contraction was maintained, blood was apparently "squeezed" proximally, and the pressure in both the deep and the superficial systems promptly returned to hydrostatic levels. At this time the valves apparently opened. On relaxation, in the presence of competent valves, the pressure fell below the hydrostatic level in segments of both the deep and the superficial systems. The return to the hydrostatic level is more rapid in the superficial femoral vein with a paucity of proximal valves than in the popliteal vein. The time required for the pressure in the deep system to return to hydrostatic levels apparently reflects the filling of valved segments of that system by inflow from the superficial system. The pressure in the saphenous vein returned to hydrostatic levels much slower than the more proximal segments, suggesting that the more proximal valves opened before the distal valves.

Subjects with the lower leg syndrome failed to demonstrate on relaxation a prolonged decrease of pressure below the hydrostatic level. This observation would suggest the absence of valvular function in these extremities.

It would appear that in normal subjects competent valves in the deep and, possibly, the superficial system effectively protect the segments of veins below, and competent communicating veins compartmentalize the deep from the superficial system. The competency of the communicating veins apparently depends upon the valves, the cross-sectional diameter of the veins, and the support afforded by the musculofascial elements. When valvular competency is lost, the venous system in the lower extremity functions as a unit.

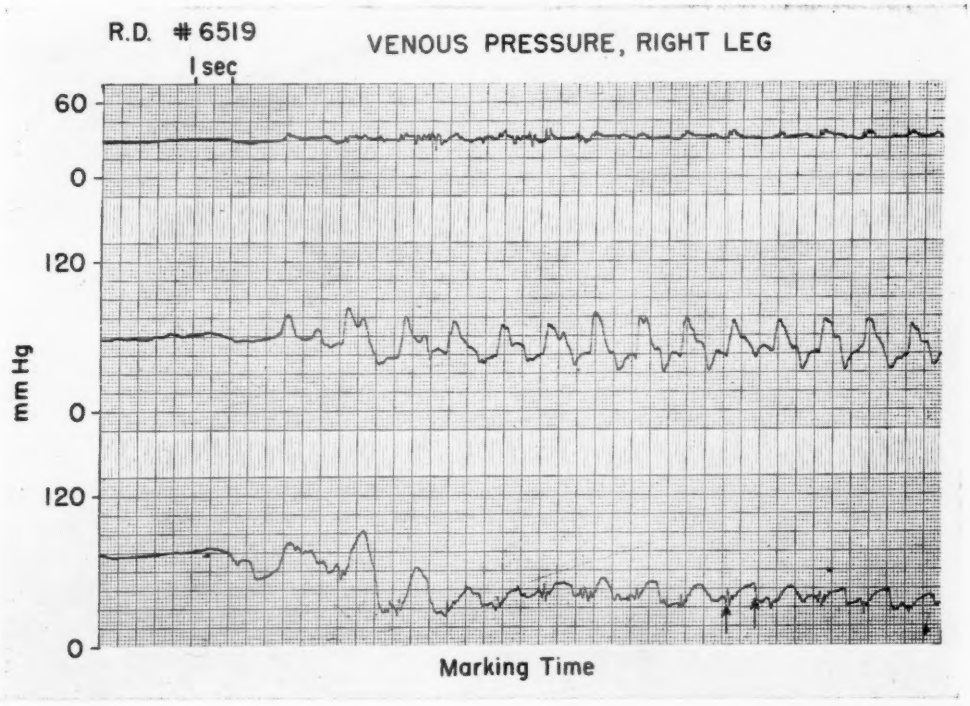


Fig. 11.—The effect of marking time, recorded from above downward in the right femoral, popliteal, and saphenous veins of a normal extremity. The first arrow indicates when the right foot touched the floor, while the second arrow marks the point where weight was borne equally by both extremities, just prior to elevation of the right foot. There is little change in femoral pressure during ambulation. During the interval of leg muscle contraction and weight bearing, the popliteal pressure is above hydrostatic and blood is propelled heartward.

Ambulation.—During ambulation, the mean pressure in the superficial femoral and popliteal veins was essentially unchanged from the hydrostatic level in both normal extremities and those with the lower leg syndrome (Table II, Figs. 11 to 13). The mean saphenous pressure decreased in normal extremities. It was unchanged or only slightly decreased in patients with the lower leg syndrome.

The decrease of saphenous pressure in this small series of normal subjects was not as marked as that reported in other studies.[7-9] The fall in pressure during ambulation is in part dependent upon the vigor of the exercise. The two or three venous catheters with attached transducers may have prevented maximum effort. The magnitude and rate of pressure fall during exercise, and the recovery after cessation of exercise, is conditioned by several factors,

TABLE II. PRESSURE OBSERVATIONS IN THE LOWER EXTREMITIES

SUBJECT	SITE	MEAN PRESSURE MM. Hg: QUIET STANDING	MEAN PRESSURE MM. Hg: MARKING TIME 30 SEC.	MEAN PRESSURE MM. Hg: CHANGE IN PRESSURE	MEAN PRESSURE MM. Hg: PER CENT CHANGE OF PRESSURE	SECONDS: CADENCE	SECONDS: MAXIMAL FALL IN PRESSURE	SECONDS: RECOVERY	RATE OF RETURN MM. Hg/SEC.	COUGH TRANSMISSION TO SAPHENOUS VEIN	REMARKS
Normal Extremities											
LS	F	27	27	0	0			0			22 yr. N.M.; R ⅓ of leg
(R)	S	75	30	−45	−60	48	2	75*		No	
FN	F	27	27	0	0			0			29 yr. W.M.; R ⅜ of thigh
(L)	S	72	36	−36	−50	48	8	†		No	
FN	F	34	38	+ 4	+12			0			Same as above; R ⅓ of leg
(R)	S	78	42	−36	−46	42	3	54	0.9	No	
HG	F	30	29	− 1	− 3			0			37 yr. W.M.; R ⅔ of thigh
(R)	S	79	45	−34	−44	54	6	20	1.7	No	
HW	F	32	32	0	0			0			35 yr. W.M.; R ⅓ of thigh
(R)	S	75	51	−24	−32	48	8	20	1.2	No	
RC	F	32	33	+ 1	+ 3						26 yr. W.M.; R ⅓ of thigh
(L)	S	81	69	−12	−15	50	10	10	1.2	No	
JE	F	31	30	− 1	− 3			0			35 yr. N.M.; R ⅜ of leg
(R)	P	42×	42	0	0			0		No	
	S	91	68	−23	−25	48	8	‡		No	
RD	F	29	30	+ 1	+ 4			0			24 yr. W.M.; R ⅓ of thigh
(R)	P	48×	40	− 8	−17			1		No	
	S	77	38	−39	−49	48	3	30	1.3	No	
RD	F	36	36	0	0						Same as above; R ⅓ of thigh
(L)	P	54	52	− 2	− 3					No	
	S	75	53	−22	−29	54	1	12	1.8	No	
HH	F	29	27	− 2	− 7						45 yr. W.M.; R ⅓ of leg
(R)	S	89	64	−25	−28	48	6	8	3.0	No	
JB	F	No ambulatory studies performed									
(R)	P									No	63 yr. W.M.; R ⅓ of thigh
	S									No	

Surgery October, 1935

						Abnormal Extremities						
WG	F	38	33	− 5	−13							75 yr. W.M.; postphlebitic; ulcer;
(L)	S	89	65	−24	−27	36	3	10	2.4	Yes	B	
HH	F	35	35	0	0							45 yr. W.M.; postphlebitic; ulcer;
(L)	S	95	95	0	0	42	0	0		Yes	B	
HG	F	31	31	0	0							37 yr. W.M.; postphlebitic; ulcer;
(L)	S	82	78	− 4	− 5	52	2	2	2.0	Yes	band R through collaterals	
HW	F	33	31	− 2	− 6							35 yr. W.M.; postphlebitic; ulcer;
(L)	S	82	66	−16	−19	54	4	4	4.0	Yes	B	
RW	F	35	33	− 2	− 6							57 yr. W.M.; trauma; no history
(R)	S	86	70	−16	−19	48	4	13	1.5	No	of phlebitis; ulcer; R ⅗ of leg	
SC	F	33	34	+ 1	+ 3							31 yr. W.M.; postphlebitic; ulcer;
(L)	P	62	62	0	0					Yes	R through recanalized superior	
	S	81	83	+ 2	+ 2	48	1	0		Yes	femoral vein	
CL	F		No ambulatory studies performed								59 yr. W.M.; trauma; no history	
(L)	S									Yes	of phlebitis; ulcer; R ⅗ of leg	

(R) Right leg; (L) Left leg; F—Femoral; S—Saphenous; P—Popliteal; B—X-ray evidence of block; R—X-ray evidence of reflux; *—Catheter directed up; †—Damped; ‡—Could not stand still for recovery; N.M.—Negro man; W.M.—white man.

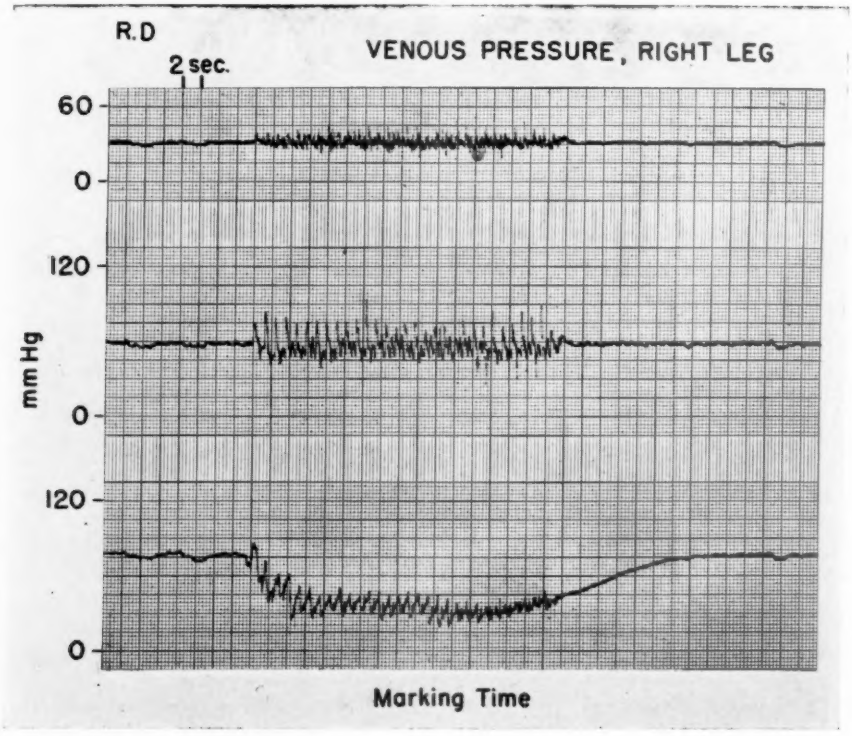


Fig. 12.—Same subject as in Fig. 11, but the pressure changes recorded at one-tenth the paper speed. Note the insignificant change in femoral pressure (upper tracing). Mean popliteal pressure (middle tracing) fell 8 mm. Hg below hydrostatic level, while the saphenous pressure (lower tracing) fell 39 mm. Hg. Observe the arterial pulse wave, present only when the pressure is hydrostatic and the valves are open.

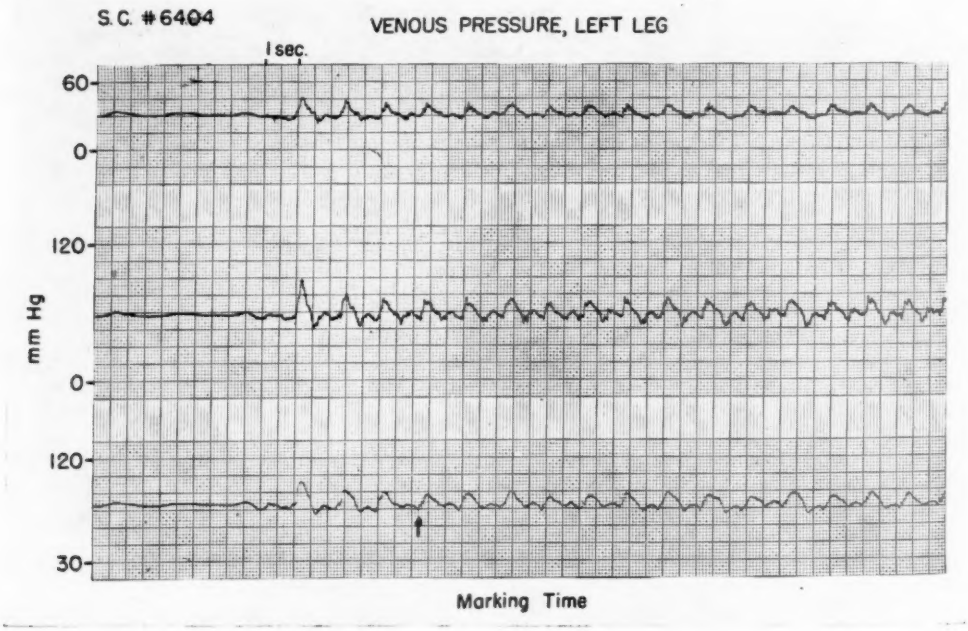


Fig. 13.—From above downward, the left femoral, popliteal, and saphenous pressures recorded from a postphlebitic extremity while marking time (same subject as Fig. 10). The arrow indicates when the left foot touches the floor. Mean pressure in the popliteal and saphenous veins does not fall significantly on ambulation. Unlike the normal subjects (Fig. 11), popliteal and saphenous pressures do not fall significantly below hydrostatic during the phase of muscular relaxation.

such as environmental temperature[16-18] and the state of the cardiovascular system.[19, 20] As these variables cannot be adequately controlled, ambulation as an isolated test of venous competency is unreliable.

Function Tests.—No single test or maneuver will adequately evaluate the state of the venous system in the lower leg. The response to cough and the Valsalva maneuver can indicate whether or not a continuous channel of venous incompetency is present. Large segments of the system may be individually evaluated by the quadriceps contraction. The response to ambulation is modified by the several variables previously noted, and may, therefore, be frequently misleading. Coupled with venographic observations, however, it would appear that utilizing all of these techniques one can usually distinguish the abnormal from the normal venous system.

DISCUSSION

Bauer[21] considered retrograde superficial femoral venography an accurate technique for evaluating valvular competency. Shumacker and associates[22] have suggested that the reflux of opaque material past valve stations could occur in normal veins if these veins became so distended that the valves were rendered incompetent. The observations of the present study suggest that the degree of reflux of opaque material does not accurately reflect the competency of the venous valve.

Fluoroscopically, such reflux in both normal and pathologic extremities was observed to occur rapidly. The valves may be momentarily shut by the pressure of the retrograde injection of contrast material which has a greater density than blood. However, in the dependent motionless extremity, these valves reopen when the gradient of centripetal flow overcomes this pressure. Successive valves might well be shut momentarily by the descending heavy opaque medium, to reopen when the pressure below this valve increased and the pressure gradient in the vein became heartward again.

Because the valves for the most part are open during retrograde venography of the motionless extremity, it appears that retrograde opacification of the deep and communicating veins is not a measure of the relative competency of the valves. Indeed, in one-third of the normal extremities, contrast material did not stop at a valve in the course of reflux. Apparently, the degree of reflux is in part determined by the rate of venous flow in the extremity. The application of heat, a relatively simple means of increasing peripheral blood flow, reduced the degree of reflux from the lower third of the thigh to the upper third in a normal extremity. The pressure patterns in the saphenous vein in the normal extremities were identical regardless of the degree of radiographic reflux in the deep system.

The venous pressure in the dependent, motionless lower extremity, with the exception previously noted, equals the hydrostatic column of blood from the level of the right auricle.[5-9] Under conditions such as quiet standing, the pressure in the venous system of normal and pathologic lower extremities exceeds the colloidal osmotic pressure of the blood. Vigorous exercise may lower the pressure in the normal superficial system so that it may become less

than the oncotic pressure of blood.[6, 23] Ambulation apparently has less effect on lowering the pressure of the deep system, however, as this system is rapidly filled by the superficial system. Accordingly, it would seem that the tendency in the capillary bed, in the erect normal subject, despite muscular activity, is toward edema formation.[24]

Counteracting this tendency is the effect of tissue pressure and lymphatic drainage. Few measurements of these factors are available. However, it may be assumed that the tissue pressure surrounding the deep system is greater than that surrounding the superficial system. The studies of Henry[16, 17] suggest that summer edema is due to the inability of muscular activity to sufficiently reduce the venous pressure in the presence of an increased blood flow associated with a warm environment. It would appear that in such a situation the lymphatics are overloaded.

The available data indicate that normally the competent communcating veins, muscles, and fascia divide the lower extremity into two vascular compartments, which are further segmentalized by competent valves. The musculofascial structures probably aid in maintaining the competency of the communicating veins. Inflammation with associated thrombosis distort the valves and communicating veins, making them incompetent.[25] Such incompetent communicating veins were a common radiographic finding in extremities with the lower leg syndrome. The venous system of the lower extremity thus becomes decompartmentalized. Under such conditions, muscular exercise is not associated with an appreciable reduction of pressure in the superficial system. Accordingly, in the upright position, the pressure in the superficial system is always above the colloidal osmotic pressure and edema develops. Inflammation would further cause blockage of lymphatic channels and favor edema formation.

Primary varicosities of the superficial veins also cause distortion of the communicating veins because of dilatation and inherent weakness, and the segmentalization and compartmentalization of the venous system disappears. Therefore, exercise is not associated with an appreciable reduction of pressure in the superficial system. The inability to lower the pressure in the superficial system during ambulation predisposes to edema formation.

Hickam and co-workers[18] have stressed the importance of chronic edema in the genesis of the lower leg syndrome. The edema may cause the mechanical disruption of cutaneous and subcutaneous structures and interfere with tissue nutrition, thereby rendering the skin less resistant to repeated infection and trauma. Such infection would lead to further lymphatic blockage and fibrosis. Thus, once the venous system has been altered, the prevention of edema in the lower extremities would appear of prime importance in prophylaxis against the development of the lower leg syndrome. This may best be accomplished by elevation of the extremity and well-applied elastic bandages.

Chronic edema is not the sole factor leading to the lower leg syndrome, however, for ulcers are uncommonly associated with the lymphedema of ele-

phantoid conditions. It is possible that venous incompetency in the lower leg permits the uninhibited transmission of frequent changes in intra-abdominal pressure. The sudden elevations of pressure transmitted through incompetent valves may well damage the surrounding tissue and predispose the extremity to poor healing and ulceration following minimal trauma.

This study supports the observations[10, 22] that ligations of the superficial femoral or popliteal veins are of no value in this disease and indeed may aggravate the process. The vigorous attack on incompetent perforators in Linton's[26] operative procedure may account for his improved results over superficial femoral vein ligation alone.

In selected patients of this series, radical excision of the ulcerated skin, subcutaneous tissue, and fascia was performed and split-thickness grafts applied in circumferential fashion to the muscle, tendon, and periosteum of the lower leg (Figs. 7*A* and 7*B*). In this manner, the superficial incompetent venous compartment with its deficient supporting tissue pressure and incompetent communicating veins was removed. Follow-up has been short but the results are encouraging thus far. A modification of this previously described procedure[27, 28] will be the subject of a subsequent communication.

SUMMARY AND CONCLUSIONS

1. A correlation of venographic observations with hemodynamic measurements in the lower extremity is presented.

2. Venographic data on 23 extremities with the lower leg syndrome is compared with similar data on 33 normal extremities. Incompetent communicating veins were a common finding in the abnormal group.

3. Venographic techniques are of unquestioned value in the demonstration of the static venous anatomy in both normal and abnormal extremities. Adequate study frequently requires both antegrade and retrograde techniques. However, venography alone, by either a retrograde or antegrade technique, provides unreliable data regarding valvular competence except in extremities with grossly abnormal communicating veins.

4. Simultaneous venous pressure measurements of the femoral, popliteal, and saphenous veins, or the femoral and saphenous veins, were recorded from 18 extremities under conditions of rest, ambulation, during cough and the Valsalva maneuver. Such studies were found to give reliable data concerning valvular competence.

5. Under conditions of quiet standing the valves of the lower extremity are open. Competent valves protect the more distal segments of the venous system from sudden increases of intra-abdominal pressure. During muscular activity, competent valves segmentalize the deep system. The valves of the communicating veins (and other, at present, poorly understood phenomena) effectively divide the leg into the two major venous compartments, the superficial and the deep system.

6. Elucidation of the genesis of the lower leg syndrome will depend upon an understanding of the normal phlebodynamics, a consideration of which must include (a) the blood flow through the extremity; (b) venomotor tone; (c) valvular competence; (d) supporting structures; and (e) other at present unknown phenomena.

REFERENCES

1. Bauer, G.: Roentgenological and Clinical Study of Sequels of Thrombosis, Acta chir. scandinav., supp. 74, **86**: 1, 1942.
2. Mahorner, H.: Indurated Leg and Leg Ulcers: A Clinical Study of Their Etiology and Treatment, Surg., Gynec. & Obst. **88**: 115, 1949.
3. Veal, J. R., and Hussey, H. H.: The Use of "Exercise Tests" in Connection With Venous Pressure Measurements for the Detection of Venous Obstruction in the Upper and Lower Extremities; a Preliminary Report, Am. Heart J. **20**: 308, 1940.
4. Winsor, T., and Burch, G. E.: Phlebostatic Axis and Phlebostatic Level: Reference Levels for Venous Pressure Measurements in Man, Proc. Soc. Exper. Biol. & Med. **58**: 165, 1945.
5. Beecher, H. K., Field, M. E., and Krogh, A.: A Method of Measuring Venous Pressure in the Human Leg During Walking, Skandinav. Arch. Physiol. **73**: 7, 1936.
6. Beecher, H. K., Field, M. E., and Krogh, A.: The Effect of Walking on the Venous Pressure at the Ankle, Skandinav. Arch Physiol. **73**: 133, 1936.
7. Warren, R., White, E. A., and Belcher, C. D.: Venous Pressures in the Saphenous System in Normal, Varicose, and Postphlebitic Extremities. Alterations Following Femoral Vein Ligation, SURGERY **26**: 435, 1949.
8. Pollock, A. A., Taylor, B. E., Myers, T. T., and Wood, E. H.: The Effect of Exercise and Body Position on the Venous Pressure at the Ankle in Patients Having Venous Valvular Defects, J. Clin. Invest. **28**: 559, 1949.
9. DeCamp, P. T., Schramel, R. J., Ray, C. J., Feibleman, N. D., Ward, J. A., and Ochsner, A.: Ambulatory Venous Pressure Determinations in Postphlebitic and Related Syndromes, SURGERY **29**: 44, 1951.
10. DeCamp, P. T., Ward, J. A., and Ochsner, A.: Ambulatory Venous Pressure Studies in Postphlebitic and Other Disease States, SURGERY **29**: 365, 1951.
11. Hojensgard, I. C., and Sturup, H.: Venous Pressure in Primary and Post-Thrombotic Varicose Veins; A Study of the Statics and Dynamics of the Venous System of the Lower Extremity Under Pathological Conditions, Acta chir. scandinav. **99**: 133, 1949.
12. Sturup, H., and Hojensgard, I. C.: Venous Pressure in the Deep Veins of the Lower Extremities of Patients With Primary and Post-Thrombotic Varicose Veins, Acta chir. scandinav. **99**: 526, 1949.
13. Tyson, M. D., and Goodlett, W. C.: Venous Pressures in Disorders of the Venous System of the Lower Extremities, SURGERY **18**: 669, 1945.
14. Liedholm, K.: Studien über das Verhalten des Venendrukes beim Valsalvashen Versuch, Acta med. scandinav., supp. 106, p. 1, 1939.
15. Gauer, O.: Personal communication.
16. Henry, J. P.: Effects of Temperature and Exercise on Venous Pressure in the Foot When in the Erect Position, Am. J. Med. **4**: 519, 1949.
17. Henry, J. P., and Gauer, O. H.: Influence of Temperature Upon Pressure in the Foot, J. Clin. Invest. **29**: 855, 1950.
18. Hickam, J. B., McCulloch, R. P., and Reeves, R. J.: Normal and Impaired Function of the Leg Veins, Am. Heart J. **37**: 1017, 1949.
19. Schneewind, J. H.: The Walking Venous Pressure Test and Its Use in Peripheral Vascular Disease, Ann. Surg. **140**: 137, 1954.
20. Edwards, W. S.: The Effect of Altering Arterial Flow on Ambulatory Venous Pressure in Post-Phlebitic Extremities, Surg., Gynec. & Obst. **99**: 756, 1954.
21. Bauer, G.: The Etiology of Leg Ulcers and Their Treatment by Resection of the Popliteal Vein, J. Internat. chir. **8**: 937, 1948.
22. Shumacker, H. B., Jr., Moore, T. C., and Campbell, J. A.: Functional Venography of the Lower Extremity, Surg., Gynec. & Obst. **98**: 257, 1954.
23. Beecher, H. K.: Adjustment of the Flow of Tissue Fluid in the Presence of Localized Sustained High Venous Pressure as Found With Varices of the Great Saphenous System During Walking, J. Clin. Invest. **16**: 755, 1937.
24. Holling, H. E., Beecher, H. K., and Linton, R. R.: Study of the Tendency to Edema Formation Associated With Incompetence of the Valves of the Communicating Veins of the Leg. Oxygen Tension of the Blood Contained in Varicose Veins, J. Clin. Invest. **17**: 555, 1938.

25. Edwards, E. A., and Edwards, J. E.: The Effect of Thrombophlebitis on the Venous Valve, Surg., Gynec. & Obst. 65: 310, 1937.
26. Linton, R. R.: The Post-Thrombotic Ulceration of the Lower Extremity: Its Etiology and Surgical Treatment, Ann. Surg. 138: 415, 1953.
27. Homans, J.: The Operative Treatment of Varicose Veins and Leg Ulcers, Based Upon a Classification of these Lesions, Surg., Gynec. & Obst. 22: 143, 1916.
28. Owens, N.: Treatment of Chronic Varicose Ulcers of Lower Extremities, New Orleans M. & S. J. 89: 483, 1937.
29. Boyce, W. H., Detar, J. H., and Vest, S. A.: A New Technique of Venography of the Lower Extremities With Urokon, Surg., Gynec. & Obst. 96: 471, 1953.

Surgical History

CONDUCTED BY JOHN R. PAINE, M.D.

THE BELLS OF EDINBURGH

JOHN W. BELL, M.D.,
SUNMOUNT, N. Y.

(From the Veterans Administration Hospital)

AS brothers, John and Charles Bell were afforded a natural kinship for collaboration early in their medical careers. Sons of a Scotch Episcopal clergyman, and through both father and mother related to several generations of ministers, neither boy was attracted to a future in the Church. John Bell, born in 1763, in Edinburgh, was Charles's elder by eleven years. Charles Bell, also born in Edinburgh, in November, 1774, was the youngest of four brothers.

The meager earnings of their father, and his death when Charles was five, gave the boys little opportunity for much formal education. Careful home tutoring by their gifted mother, particularly for Charles after the other brothers had left home, resulted in a background of self-reliance and general knowledge equal to any of their contemporaries.

The basis for John Bell's decision to study medicine is unknown. No direct family influence is evident other than an intellectual inheritance and artistic spirit. Edinburgh University of that day had a remarkable collection of outstanding thinkers and was a true medical fountainhead. Perhaps the challenge of this environment was the spark for John Bell's interest in anatomy and surgery. At any rate, it appears that Charles was directly influenced by his brother's early accomplishments. John, by 1790 a young faculty member, was in a position to teach Charles the elements of anatomy and surgery.

Both brothers inherited an unusual ability to draw and were soon medical artists in their own right. This talent, fostered by their mother, was further coached by David Allan, an artist at the University. The fruition of this medical school association was apparent when, in 1799, shortly before graduation, Charles Bell published his *System of Dissections,* with plates engraved from his own sketches, and wrote the description of the nervous system in John Bell's *Anatomy of the Human Body* (first published in 1793).

The practical spirit of British anatomists, which prevailed toward the close of the eighteenth century, is exemplified in the character and work of John Bell.

> In the latter part of that century, British anatomists were, as indeed they had always been, much more than mere describers. John Hunter, in London, and Alexander Monro (secundus), in Edinburgh, had modelled themselves on the prototype of English anatomist, William Harvey. In no sense was

> Harvey a descriptive anatomist: he studied the human body to understand the significance, not the form, of the various parts. He perceived that the hypotheses of his time did not account for the structure of the heart nor for the arrangement of its blood vessels. It was to explain these that he postulated the theory of the circulation of the blood, the truth of which he afterward proved by experiment. It was in the same spirit that Hunter, Monro, and the two Bells pursued the study of anatomy in England in the latter decades of the eighteenth century.[1]

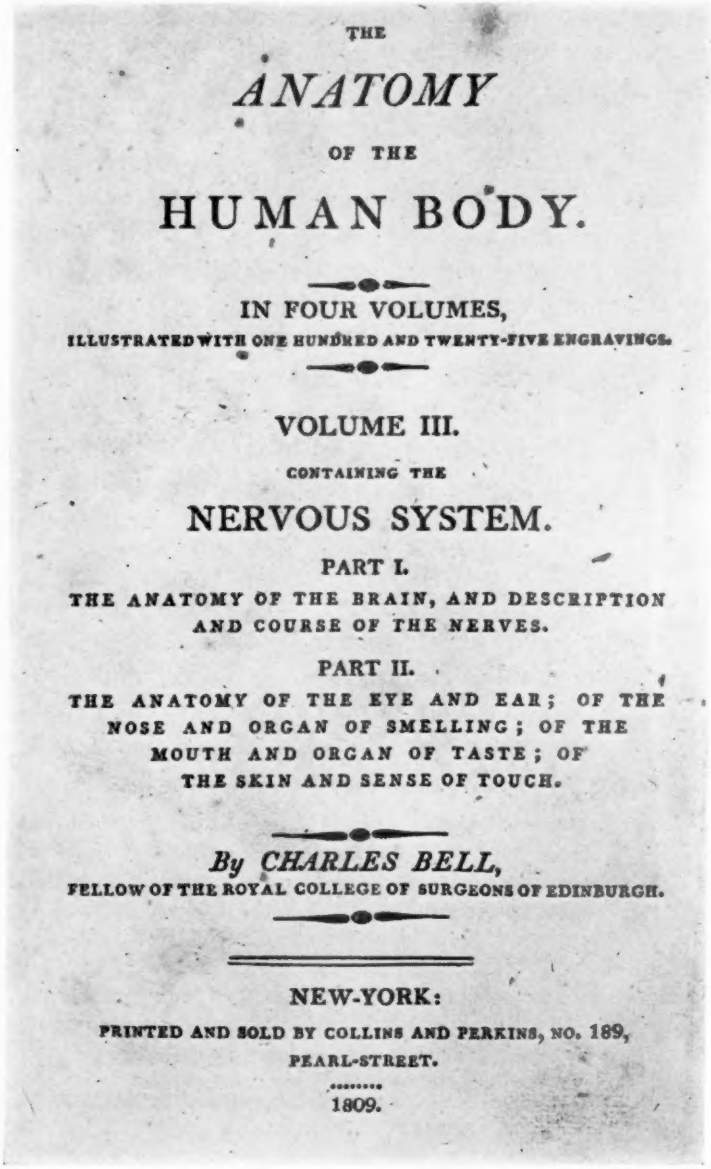
Although the celebrated Alexander Monro (secundus), John Bell's senior in the Department of Anatomy, had physiologic insight in the same sense as William Harvey, having formed a true conception of the manner in which the cerebral circulation was carried on from a consideration of the anatomy of the parts, Monro tertius was caught up in the vogue of the expository lecture created by Marie Bichat in Paris. In particular, the "evergreen tertius," up to 1846, "unconcernedly at noon, ate cranberry tarts in the midst of grinning students at a small pastry cook's and with digestion unimpaired the next hour read his grandfather's essays on hydrophobia as part of the anatomical course." John Bell, in early resentment of these ineptitudes of "the windy and wordy" school, opened a private school of anatomy in 1790. He also pointed out that "in Dr. Monro's class, unless there be a fortunate succession of bloody murders, not three subjects are dissected in the year, while nerves and arteries which the surgeon has to dissect at the peril of his patient's life were demonstrated on a subject fished up from the bottom of a tub of spirits and exhibited at a distance of a hundred feet."

After graduation in August, 1799, Charles Bell was admitted to the Edinburgh College of Surgeons and, subsequently, to the surgical staff of the Royal Infirmary, the only hospital in Edinburgh. Soon after this appointment, there arose a sharp feud between the older and younger staff members. Possessed of a combative temperament, John Bell continued to rail at the mistakes of Benjamin Bell (no relation) and the Monros.

> The older members, led by James Gregory, the internist, attempted to oust the younger men who were championed by John Bell. This soon crystallized into a bitter and acrimonious dispute between Gregory and Bell. Gregory, apparently not having sufficient wit to use the rapier, wielded the bludgeon very heavily. 'Any man,' he said, 'if himself or his family were sick, should as soon think of calling in a mad dog as Mr. John Bell.' The latter retorted with a weapon equally blunt, accusing Gregory of being motivated by selfish and base desires. But Gregory was in a position of greater power: John and Charles Bell were not reappointed to the infirmary.[2]

Returning to his school of anatomy, John Bell pursued his careful and honest investigations. It is not clear where he carried on his surgical practice, since the scheming of Gregory prevented his use of the Royal Infirmary. He became interested in vascular surgery and successfully tied the common carotid and posterior branch of the internal iliac and was the first to ligate the gluteal artery. Garrison[3] considered him, with Desault and Hunter, a founder of the modern surgery of the vascular system.

John Bell's *Anatomy of the Human Body* (1793-1803) was issued in three volumes with almost all of the drawings, etchings, and engravings being his own, while the third volume, dealing with the brain, nerves, sense organs, and viscera (1804) was almost entirely the work of Charles (Fig. 1). This book was important not only as a milestone in the history of anatomic delineation but also because it represented the last truly British textbook of anatomy before 1900.[1]



THE

ANATOMY

OF THE

HUMAN BODY.

IN FOUR VOLUMES,

ILLUSTRATED WITH ONE HUNDRED AND TWENTY-FIVE ENGRAVINGS.

VOLUME III.

CONTAINING THE

NERVOUS SYSTEM.

PART I.

THE ANATOMY OF THE BRAIN, AND DESCRIPTION AND COURSE OF THE NERVES.

PART II.

THE ANATOMY OF THE EYE AND EAR; OF THE NOSE AND ORGAN OF SMELLING; OF THE MOUTH AND ORGAN OF TASTE; OF THE SKIN AND SENSE OF TOUCH.

By CHARLES BELL,

FELLOW OF THE ROYAL COLLEGE OF SURGEONS OF EDINBURGH.

NEW-YORK:

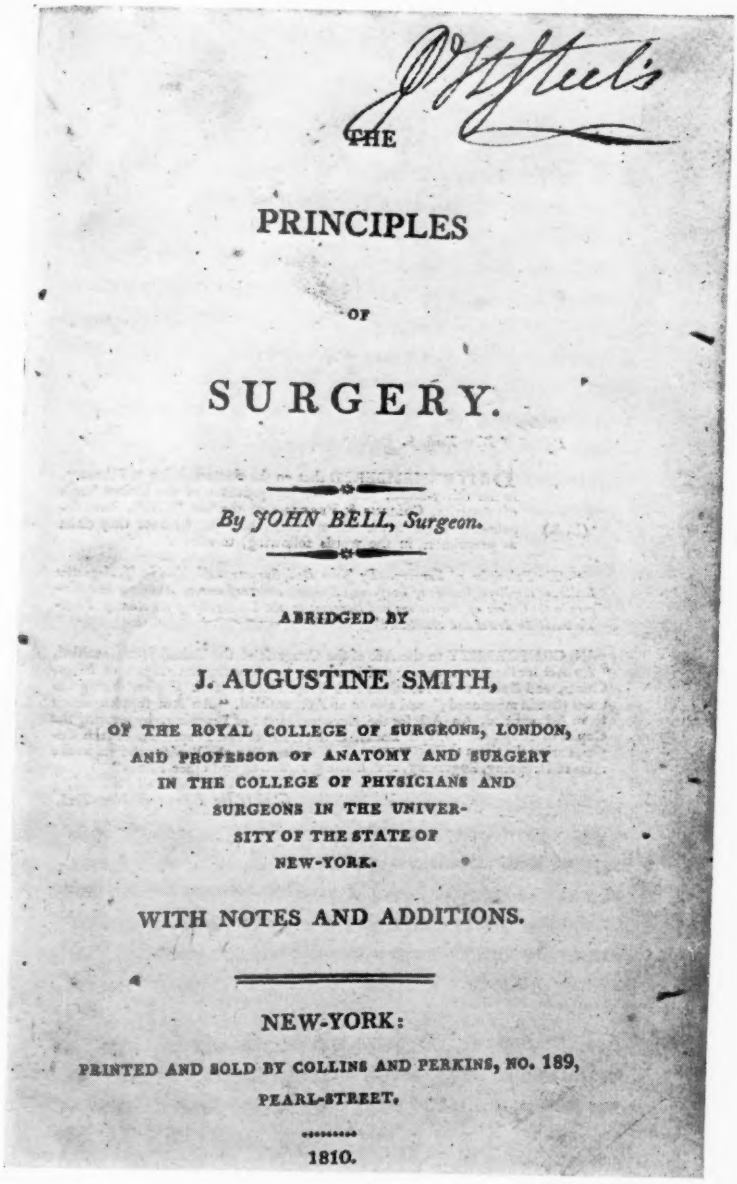
PRINTED AND SOLD BY COLLINS AND PERKINS, NO. 189, PEARL-STREET.

1809.

Fig. 1.

Completely uninfluenced, indeed contemptuous of the purely descriptive school, surgeon-anatomist John Bell wrote with great sincerity and an effort at physiologic interpretation. John Bell's most enduring contributions to surgery

are his *Discourses on the Nature and Cure of Wounds* (1795), the second of which is a valuable discussion of the surgery of the arteries; and his monumental *Principles of Surgery* (1801-1807), embellished with beautiful original engravings and full of unique historical and clinical matter relating to the ligation of the great vessels, fractures, trephining, tumors, and lithotomy, of which he gives a detailed history, occupying 248 pages (Figs. 2 and 3).



THE

PRINCIPLES

OF

SURGERY.

By JOHN BELL, Surgeon.

ABRIDGED BY

J. AUGUSTINE SMITH,

OF THE ROYAL COLLEGE OF SURGEONS, LONDON,
AND PROFESSOR OF ANATOMY AND SURGERY
IN THE COLLEGE OF PHYSICIANS AND
SURGEONS IN THE UNIVER-
SITY OF THE STATE OF
NEW-YORK.

WITH NOTES AND ADDITIONS.

NEW-YORK:

PRINTED AND SOLD BY COLLINS AND PERKINS, NO. 189,
PEARL-STREET.

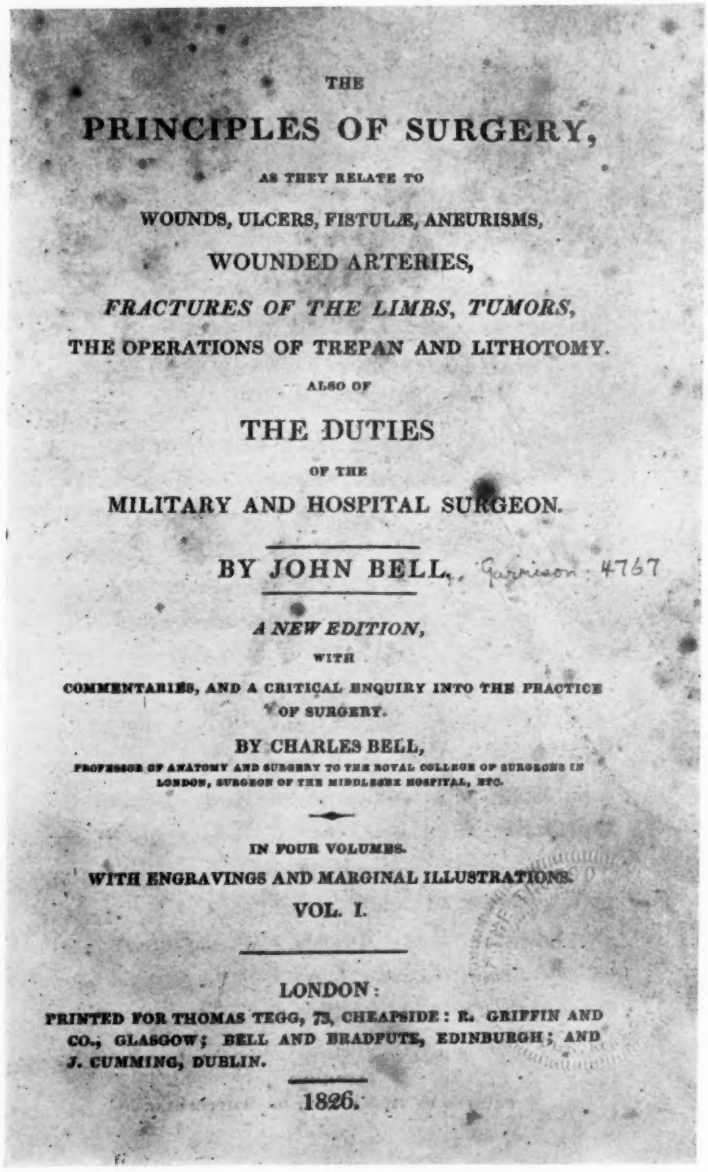
1810.

Fig. 2.

Toward the end of his life, broken in health by a fall from a horse, John Bell went to Italy. Long an enthusiastic admirer of this country, he died here in 1820. He left a posthumous book entitled *Observations on Italy* (1825),

which is remarkable for its beautiful original drawings, vivacity of style, and extensive knowledge of Italian art. Garrison regarded it as one of the best books of travel ever written by a physician.

After James Gregory had so rudely closed the door in his face, Charles Bell decided that Edinburgh afforded him little chance for advancement and went to London in 1804 to resume his studies and to practice there.



THE

PRINCIPLES OF SURGERY,

AS THEY RELATE TO

WOUNDS, ULCERS, FISTULÆ, ANEURISMS,

WOUNDED ARTERIES,

FRACTURES OF THE LIMBS, TUMORS,

THE OPERATIONS OF TREPAN AND LITHOTOMY.

ALSO OF

THE DUTIES

OF THE

MILITARY AND HOSPITAL SURGEON.

BY JOHN BELL. Garrison 4767

A NEW EDITION,

WITH

COMMENTARIES, AND A CRITICAL ENQUIRY INTO THE PRACTICE OF SURGERY.

BY CHARLES BELL,

PROFESSOR OF ANATOMY AND SURGERY TO THE ROYAL COLLEGE OF SURGEONS IN LONDON, SURGEON OF THE MIDDLESEX HOSPITAL, ETC.

IN FOUR VOLUMES.

WITH ENGRAVINGS AND MARGINAL ILLUSTRATIONS.

VOL. I.

LONDON:

PRINTED FOR THOMAS TEGG, 73, CHEAPSIDE: R. GRIFFIN AND CO., GLASGOW; BELL AND BRADFUTE, EDINBURGH; AND J. CUMMING, DUBLIN.

1826.

Fig. 3.

Medical circles in London were equally hostile to young Charles Bell. After several months, only one friend, Lynn, stood up to his critics with, "I like his brother and I like himself. He is no humbug; his conversation is open and free."

The first real encouragement for Charles was a lukewarm invitation to join the Anatomical Society. Having heard of him through his *Dissections* and his chapters in John Bell's *Anatomy,* this group, including Cline, Cooper, Abernethy, Home, Wilson, Baillie, and Blizzard, condescended to his membership. Presented with similar barriers in London to those experienced earlier by John in Edinburgh and unable to find an opening in an established institution, Bell decided to found one of his own. So, on Jan. 20, 1806, he took a house on Leicester Street, fitted it up as a lecture room and living quarters, and established himself as a lecturer.

Though actual income was slow, the lectures were an unqualified success. His fame spread rapidly and, in 1812, Wilson offered him a partnership in the Great Windmill Street Medical School. The school had been founded by the Hunters and had been the scene of their great success. Charles was fond of modeling pathologic specimens in wax and, upon moving to Windmill Street, his anatomic and pathologic preparations were incorporated in the Hunterian collection. Bell's enthusiasm for his new position was reflected in the inspiration of his brilliant lectures.

In addition to his popularity with medical students and visiting physicians, Bell also lectured to artists, his *Anatomy of Expression* (1806) being the result of these studies.

Interested in the nervous system since his undergraduate days in Edinburgh, Charles wrote in a letter to his brother, George, dated Nov. 26, 1807, "I have done a more interesting nova anatomia cerebri than it is possible to conceive. I lectured it yesterday. I prosecuted it until 1 o'clock, and I am sure that it will be well received."[4]

In 1811, he privately printed 100 copies of a pamphlet entitled, "Idea of a New Anatomy of the Brain." Since this paper was distributed only to the author's private friends, the work was unknown to the scientific world.

In Bell's own words, he was attempting in the "Idea" to combat the "prevailing doctrine of the anatomical schools . . . that the whole brain is a common sensorium . . . and that the mind, by the same nerves which receive sensation, sends out the mandate of the will to the moving parts of the body"; and to prove "that the cerebrum and cerebellum are different in function as in form; that parts of the cerebrum have different function; and that the nerves which we trace in the body are not single nerves whose filaments are united for the convenience of distribution, but which are distinct in office, as they are in origin in the brain. . . .

"That the nerves of sense, the nerves of motion, and the vital nerves are distinct through their whole course, though they seem sometimes united in one bundle; and that they depend for their attributes on the organs of the brain to which they are severally attached."

Bell further defined "vital nerves": "The secret operations of the vital organs suffer the control of the brain, though we are unconscious of the thousand delicate operations which are every instant going on in the body." It is apparent that by this he meant what we now term the autonomic nervous control of the viscera and other organs.

Since, in Bell's opinion, it was difficult, if not impossible, to experiment directly on the brain itself, he thought he could "touch" the cerebrum by stimulation of the anterior fasciculus of the cord, and the cerebellum by stimulation of the posterior fasciculus.

Attempting experimental proof in the spinal cord of an ass, he found, "I could cut across the posterior fasciculus without convulsing the muscles of the back; but that on touching the anterior fasciculus with the point of the knife, the muscles of the back were immediately convulsed."

J. M. D. Olmsted[5] summarized Charles Bell's famous "Idea" thus: the cerebrum is the center for both sensation and motion, the pathway for both these functions is over the ventral columns of the spinal cord and the ventral roots of the spinal nerves; the cerebellum is the center for unconscious impressions from the body itself and involuntary movements, and their pathway is over the dorsal columns of the spinal cord and the dorsal roots of the spinal nerves.

Bell's guess as to the specific function of the dorsal and ventral roots was no nearer the truth than that of Alexander Walker, who seems to have been the first actually to propose that one set of spinal nerve roots serves for sensation, the other for motion. Walker's suggestion was published in the Archives of Universal Science in July, 1809, two years before the printing of Bell's pamphlet, but Walker ascribed sensation to the ventral roots and motion to the dorsal roots.[5]

When Magendie, in 1822, began his series of experiments on a litter of eight puppies which demonstrated that the dorsal roots were "mainly for sensation" and the ventral roots "mainly for motion," this was entirely new light on the question and Bell at once revised his earlier conjecture.[6]

In 1824, he republished certain of his papers, previously read before the Royal Society, under the title, *An Exposition of the Natural System of the Nerves of the Human Body,* prefacing them with a short introductory essay in which he outlined his revised concept as to the location of the different nerve centers. He now considered the dorsal roots to be purely sensory, and because of their supposed connections with the cerebellum via the dorsal columns of the spinal cord, the cerebellum must be the seat of sensation; the ventral roots now became purely motor to him, and since they were connected with the cerebrum via the ventral columns, the cerebrum became the center for motion.

Bell's concepts of neuroanatomy and physiology prevailed, however, until 1855. During this period a great number of investigators, following the lead of Magendie, tried their hand at cutting the various spinal columns, repeatedly at different levels. Roland, Turk, Longet, and others added their support and refutation to the controversy.

It remained for C. E. Brown-Séquard,[1] after nine years of careful and determined experimentation, to prove to the satisfaction of the Paris Société de Biologie in July, 1855, that hemisection of the animal cord resulted in dis-

appearance of sensation on the opposite side of the body below the lesion and motor paralysis on the same side. This was the final push which overthrew Bell's "Idea."

It was unfortunate that anatomic methods of that era did not permit Bell and others to determine the validity of their concepts. Only after the introduction of Waller's method of tracing nervous tracts by degeneration, and the Marchi method of staining, did our knowledge of pathways in the central nervous system become precise.

In 1813, Charles Bell believed that strabismus could be relieved surgically by cutting the nerve to the intact muscle responsible for the eyeball deviation. Pursuing this idea, Charles had his assistant, John Shaw, look all over London for a cross-eyed monkey. In default of a squinting monkey, Bell decided to try section of the facial nerve. DuBois D'Amiens described the event as follows:

> The poor animal began to grimace with redoubled energy, but entirely on one side of his visage, the other remaining quite motionless. No one, assuredly, could have thought of practicing this experiment upon a human being, but nature took it upon herself.
>
> All who were admitted to see the monkey operated on by Charles Bell were struck with the strange analogy its physiognomy presented with that of an old actor, then high in repute in London, who could at pleasure express every variety of passion on one side of his face, while the opposite half exhibited no emotion whatever. Charles Bell's experiment supplied him with a clue to the enigma. It was found that this comedian, struck by facial hemiplegy, in consequence of an accidental injury to the motor nerve, simply availed himself of a natural infirmity.

Thus the eponym, Bell's Palsy, had its origin and subsequently has been applied to paresis resulting from a peripheral lesion of the facial nerve.[2, 7]

During this period, honors came rapidly to Charles Bell. He was admitted to the Royal College of Surgeons in 1813 and the following year was elected surgeon to the Middlesex Hospital.

In spite of a burgeoning practice, he managed to find time for writing and sketching. In 1807, his *Operative Surgery* appeared, and, in 1810, *Diseases of the Urethra.* Continued activity in surgical pathology resulted in *Engravings From Specimens of Morbid Parts,* published in 1813.

The battle of Coruna in 1805, and later Waterloo in 1815, found Charles Bell anxious to help, and with John Shaw he left for the battlefield to treat the wounded. A skillful and rapid operator, Bell possessed originality and dispatch in his surgical decisions. Battlefront surgery, however, taxed his almost inexhaustible powers and for days he operated continuously from six o'clock in the morning until seven in the evening. Of these experiences, he wrote,

> It was a strange thing to feel my clothes stiff with blood and my arms powerless with exertion of using the knife; and more extraordinary still, to find my mind calm amidst such variety of suffering; but to give one of these objects access to your feelings was to allow yourself to be unmanned for the performance of duty.

Returning to London, he summarized his experience in a work on gunshot wounds. Then appeared in rapid succession his *Surgical Observations, Essay on the Forces Which Circulate the Blood,* and *Observations on Injuries of the Spine.* A paper describing the external respiratory nerve (Bell's nerve) to the serratus anterior muscle appeared in 1821.

Two years later, arising out of his interest in the extraocular muscles and the seventh cranial nerve, Charles Bell described the changed ocular rotation in facial palsy—Bell's phenomenon. A related observation accredited to Charles was his report that the trigeminal nerve was both motor and sensory.

Fig. 4.—Sir Charles Bell. (From Garrison,[3] with permission of W. B. Saunders Company.)

These discoveries and his widespread fame led to his appointment as Professor of Anatomy in the Royal College of Surgeons in 1824. His lectures were crowded to standing room only, and wealthy patients sought him out for consultation. During these pleasant days, at the height of his career, Charles Bell was a genial, unaffected, kindhearted man, with a captivating twinkle

behind his eyeglasses, a bit of a dandy in attire (Fig. 4). The highest accolade came in 1831 when the order of Guelphic Knighthood was conferred upon him by the enthusiastic Lord Brougham for his outstanding work in physiology.

After John Bell had left for Italy some 20 years before, the opposition to the Bells in Edinburgh had been forgotten. Anxious to share in the fame of Sir Charles Bell, the Edinburgh Council, in 1835, offered him the chair of Surgery at the University. This change was not unwelcome to Sir Charles. His heart was in Scotland, and he longed for the streets of Edinburgh. Since two older brothers, Robert and John, had died several years before, Sir Charles yearned for the companionship of his remaining brother, George, Professor of Law at the University. "London," he wrote, "is a place to live in, but not to die in. My comfort has ever been a labour for some great purpose, and my great object of study has been attained. There is but one place where I can hope to fulfill the object of my scientific labour, and that is in Edinburgh."

Sir Charles's lifelong effort to interpret the form of a part in relation to its function reached its culmination in his concept of *The Hand, Its Mechanism and Vital Endowments, Envincing Design* (first published 1837, seventh edition revised by Alexander Shaw, London, 1865).[8] Mature reflection, based on a strong religious heritage, stimulated Charles Bell to consider the hand as an object designed by the Eternal Architect. The argument of design, which occupied the minds of natural philosophers for centuries, persisted as a doctrine of natural theology in Bell's time, which emphasized evidences of contrivances and design in nature ordered by an Intelligent Being for a purpose.

Sir Charles had already presented a discussion of the concept of natural theology in his treatise on "Animal Mechanics" (1827). In fact, some eighteen years before (1815), in one of his many preserved letters[4] to his brother, is found the reference,

> I, for my part, have no pleasure but in anatomy. You will say that it is that I may become the captain of anatomists; but why then have I such inexhaustible delight in the whole *face of nature?* No, it is in the pleasure I have in investigating structure. Everything there so *perfect,* so curiously *fitted* and leading you, little by little, to the comprehension of a Wisdom.

In the Introduction to *The Hand,* Bell takes the position that in the mechanical construction of animals, as in their endowments of life, they are created *in relation to the whole,* planned together and fashioned by one MIND and, in addition, that nothing is satisfactory until it is declared and believed that it has been the will of an Omnipotent Being to create.

We may rightly ask the question as to why Bell found the human hand a favorable field for his intellectual powers. The answer again is to be found in his own words, "the hand represents the last and best proof in the order of creation of that principle of Adaptation which evinces Design." Further, "there is extreme grandeur in the thought of anticipating or prospective Intelligence, nor are these conclusions too vast to be drawn from the examination of a part so small as the Human Hand."

To support his theses in *The Hand,* Bell, the scientist, goes to the field of Comparative Anatomy to bring forth evidence of perfection of structure, and he uses this tool because of his respect for the genius and good sense of the distinguished naturalist, Cuvier.[9]

In his discussion of "Sensibility and Touch," Sir Charles Bell makes the keen observation and develops the thought that in the matter of the sense of touch, we must not only feel the contact of the object, but we must be sensible of the *muscular effort* made to reach or grasp it in the fingers. To this consciousness of muscular exertion, he gives the name "muscular sense," calling it a sixth sense.

In this connection, Leonard Carmichael[10] quoted the great neurophysiologist, Charles Sherrington, to the effect that Sir Charles Bell was the first to postulate definitely the modern physiologic doctrine of the muscle sense and to place this modality on a scientific parity with the five senses of antiquity.

This treatise on *The Hand* was chosen by the President of the Royal Society as one of eight similar philosophical essays which satisfied the terms of the will of the Eighth Earl of Bridgewater. The Earl, the Reverend Francis H. Egerton, who died in the year 1829, specified that 8,000 pounds was to be paid to the author of the best treatise "On the Power, Wisdom and Goodness of God as manifested in the Creation."

Rather than select one essay, the executor of the will believed the ramifications of this definition were such that the money should be divided equally among eight men. Thus, Sir Charles Bell received 1,000 pounds and the copyright of his own treatise. (Bridgewater Treatise, "The Hand," 1837).

During these latter years, Sir Charles Bell continued his output of scientific studies in anatomy and physiology.[11] Among his other works, which were illustrated with his own sketches, may be mentioned, *An Exposition of the Natural Nerves of the Human Body* (1824); *The Nervous System of the Human Body* (1830); and many papers in the Medico-Chirurgical Transactions of the Royal Society. (Medical Classics, October, 1936, lists 143 items in his bibliography.)

Sir Charles had been happily married for 31 years, but there is no record of any children from this union. In April, 1842, a rumor reached his ears that the government was going to abolish the subsidy to Edinburgh University, and he hastened to London, with Lady Bell, to argue against the abolition of his beloved school. On the way, while at Manchester assisting at an operation, he was attacked with such severe chest pain that he thought his end had surely arrived. Sensing, as did John Hunter, that his heart was warning of impending disaster, he nevertheless continued his journey. Suffering from several paroxysms of angina the next day, he maintained outward calm to keep from worrying his wife and friends.

> Before bedtime he read outloud the twenty-third psalm, then retired and fell into a deep, quiet sleep. In the morning he awoke with severe pain, which he said was due to changing his position. His wife arose to give him some laudanum. Calling her to him, he laid his head on her shoulder and passed away.[2]

No more fitting tribute has been paid to Sir Charles Bell than that in the Edinburgh Review for April, 1842:

> Never passed away a gentler, truer, or finer spirit. His genius was great and has left a legacy to mankind, which will keep his name fresh in many generations. But the story of his life was a more potent moral. It is the story of one who kept his affections young, and his love of the pure unsullied, while fighting bravely the battle of life; whose heart was as tender as his intellect was vigorous and original, who, while he gained a foremost place amongst his fellows, turned with undiminished zest to his home and his friends, and found there the object, the reward and the solace of his life.

Sir Charles was buried near a yew tree that he had sketched a few days before, in Hallow Churchyard. At first a plain stone marked the spot, with his name, dates of birth and death, and the line, "The pure in heart shall see God." Afterward a tablet was placed in the Churchyard with an inscription by his lifelong friend and schoolmate, Francis (Lord) Jeffrey. It reads, "Sacred to the memory of Sir Charles Bell, who, after unfolding with unrivaled sagacity, patience, and success, the wonderful structure of our mortal bodies, esteemed lightly of his greatest discoveries, except only as they tended to impress himself and others with a deeper sense of the infinite wisdom and ineffable goodness of the Almighty Creator."

REFERENCES

1. Keith, Arthur: Human Anatomy in England During the Nineteenth Century, Lancet (Jan.), 1908.
2. Paskind, Harry A.. Sir Charles Bell: A Biographical Sketch, Society of Medical History of Chicago, vol. V (Jan.), 1937, pp. 34-43.
3. Garrison, F. H.: Introduction to the History of Medicine, ed. 4, Philadelphia, 1929, W. B. Saunders Company, p. 173.
4. Letters of Sir Charles Bell, Selected From His Correspondence With His Brother, George Joseph Bell, 1870 (Vol. of Letters, Published by Lady Bell, London), p. 180.
5. Olmsted, J. M. D.: The Aftermath of Charles Bell's Famous "Idea," Bull. Hist. of Med. **14:** 341, 1943.
6. Keith, Arthur: Correspondence, Letters to Editor, Lancet **VI,** p. 764, 1911.
7. Kelly, E. C.: Bell's Palsy, Medical Classics, vol. I, 1936-1937, p. 81.
8. Bell, Sir Charles: The Hand, Its Mechanism and Vital Endowments, Envincing Design, Revised by Alexander Shaw, ed. 7, London, 1865, p. 113.
9. Spector, Benjamin: Sir Charles Bell and The Bridgewater Treatise, Bull. Hist. of Med. **12:** 314, 1942.
10. Carmichael, Leonard: Sir Charles Bell—A Contribution to the History of Physiological Psychology, Psychol. Rev. **33:** 204, 1926.
11. Experimental Ligation of the Arteries and Nerves in Dogs, Guy's Hosp. Rep. **1:** 457: 654, 1836.
12. Castiglioni, A.: A History of Medicine, ed. 2, New York, 1947, Alfred A. Knopf, Inc.

Appraisal of Progress in Surgical Therapy

CONDUCTED BY WARREN H. COLE, M.D.
ASSISTED BY
J. GARROTT ALLEN, M.D. CLARENCE DENNIS, M.D.

THE TREATMENT OF BURNS

CARL A. MOYER, M.D., ST. LOUIS, MO.

(From the Department of Surgery, Washington University School of Medicine)

THE degree of improvement in the therapy of thermal injuries can be gauged in part by comparing the mortality attending thermal injuries in the past and present. Table I compares the death rates of serious burns thirty years ago and today.

The mortality rate attending mixed second and third degree burns covering 0 to 25 per cent of the body is not significantly lower today than it was thirty years ago. The same thing is true for injuries covering more than 75 per cent of the body's surface. In other words, from the mortality standpoint, there has been little improvement in the medical handling of burns covering less than 25 per cent and more than 75 per cent of the body during the past quarter century. However, there has been significant improvement in the treatment of burns covering 25 to 75 per cent of the body's surface.

Why have the mortality rates of burns covering 25 to 75 per cent declined while the rates for those covering less than 25 per cent and more than 75 per cent have not?

This question cannot be answered definitely; however, it may be approached by examining the peculiarities of the problems posed by thermal injuries covering 0 to 25, 25 to 75, and 75 to 100 per cent of the body.

The major physiologic and surgical problems posed by thermal injuries are: (1) peripheral circulatory failure; (2) infection; (3) biochemical disturbances; (4) nutritional disturbances; (5) mental disturbances; (6) the restoration of the cutaneous covering.

Mixed second and third degree thermal injuries covering 0 to 25 per cent of the body of all but infants and aged patients are rarely associated with lethal shock even though the injured person be given nothing more than ordinary food and drink (Table II). The few who develop shock are readily treated with a solution containing sodium salts such as normal saline, Hartmann's solution, plasma, or serum. These solutions are effective when given orally to those who are not nauseated or vomiting.

Save for the rather rare surgical erysipelas and tetanus, infections have not posed particularly difficult problems with these relatively small burns since the application of aseptic techniques in the care of the thermal wound.

Received for publication Aug. 16, 1955.

TABLE I. MORTALITY OF BURNS AMONG PERSONS AGED LESS THAN 61 YEARS

AREA INJURED PER CENT BODY SURFACE SECOND AND THIRD DEGREE	MORTALITY PER CENT			
	1920-1930*	1946-1953†	NO. OF PATIENTS	DIFFERENCE
15-24	7	5	173	2.0
25-34	30	6	66	24.0
35-44	74	36	50	38.0
45-54	89	58	26	31.0
55-64	94	78	27	16.0
65-74	100	86	15	14.0
75-84	100	91	11	9.0
85-94	100	100	9	0.0
95-100	100	100	5	0.0

*Derived from probit curve based on data of Riehl, G., Jr. (Arch. Dermat. u. Syph. 153: 41, 1927).
†Actual death rates among 382 thermal injuries in Dallas and St. Louis, 1946-1953.

Life-endangering biochemical, nutritional, and mental disturbances attending burns of less than 25 per cent of the body are relatively few and are largely self-correcting, provided the individual eats at all and has the areas of full-thickness injury covered by split-thickness grafts before the fiftieth postburn day.

In brief, thirty years ago the therapeutic problems posed by burns of less than 25 per cent of the body could be met effectively without antibiotics, without the transfusion of blood or plasma, without special nutritional care, and without an extensive knowledge of fluid balance and endocrines.

TABLE II. SHOCK THERAPY, DALLAS AND ST. LOUIS
(AGES 3 MONTHS TO 61 YEARS, 48 HOURS POSTBURN, SECOND AND THIRD DEGREE)

PER CENT BODY SURFACE INJURY	THERAPY			
	NONE (NO SALINE) (NO BLOOD) (NO COLLOID)	BUFFERED SALINE	COLLOID BLOOD OR PLASMA, OR BOTH	
8-14	58*	14	8	
15-19	40	19	6	
20-36	17	23	24*	
Totals	115	56	38	209
*Deaths	1	0	1	2
Cause of death	Tetanus		Tension pneumothorax	

The fact that the addition of antibiotics, the transfusion of blood and plasma, the application of the principles of fluid balance, and a greatly expanded knowledge of the metabolic alterations attending thermal injury have not significantly reduced the mortality of burns covering less than 25 per cent of the body from what it was thirty years ago when asepsis, early skin grafting, occasional saline infusions, and regular diets constituted the sole basis of therapy is sufficient to permit the statement: The primary requisites for the treatment of relatively small burns are the alleviation of pain when it is suffered, the aseptic handling of the wound, and early skin grafting.

The mortality rate associated with burns of more than 75 per cent of the body's surface of mixed second and third degree burns has not changed materially during the last three centuries; it was 100 per cent in Paré's time

and it is still close to that today. This is so because we are still incapable of coping with the enormity of the problems posed by such injuries. Although death from shock is now relatively rare in this category of burns, the subsequent nutritional, bacterial, psychopathologic, and cutaneous coverage problems are as yet insurmountable. More investigations of the peculiarities of the anabolic and catabolic deviations associated with burns, of the nature of the function of skin as an organ, of methods of controlling infection, and of the nature and methods of controlling burn-psychosis are needed before much improvement can be expected in the treatment of the very extensively burned person. Cortisone is not the answer.

Although we cannot brag of our accomplishments in the treatment of small or massive burns, we can do so in the case of burns covering 25 to 75 per cent of the body; the advance has been remarkable considering our relatively fragmentary knowledge of the pathophysiology of thermal injury. The problems posed by this category of burns are great but can be met by the intelligent application of what little is known.

The transfusion of blood coincident with or following the giving of solutions of sodium salts or plasma has effected easier and surer shock therapy than was attainable with the giving of blood, of saline solutions, or of plasma alone. The destruction of the element responsible for "serum-jaundice" by the storage of plasma at room temperatures for six months permits the use of pooled plasma with safety.[1]

The status of dextran in the treatment of burn-shock is still indeterminate. There is no evidence that it is more effective than a buffered saline solution in burn-shock treatment. However, there is evidence that some dextrans are not as effective as an isotonic saline solution in treating burn-shock. The dextran does not stay in the circulation of a burned animal; it enters the injured part as do the proteins or plasma. More objective research into the relative merits of plasma, saline solutions, and dextran in treating burn-shock is sorely needed.

Death of the burned from surgical erysipelas, pneumonia, and tetanus is very rare now. The antibiotics and tetanus toxoid are largely responsible for this. Although death from streptococcic and pneumococcic infections is rare, the control of staphylococcic infections is becoming more difficult; the "hospital" strains are becoming progressively more resistant to antibiotics. Pseudomonas infections are as resistant to treatment as they always have been. The exposure of the wound to air after repeated cleansing with frequently changed saline-wet lay-on dressings is the most effective means of handling them.

Progress in the management of burn-psychosis has been nil. Without doubt, it alone is responsible for a great deal of the morbidity and some of the death rate. Evidently surgeons must become psychiatrists in order that the psychiatric problems of surgery be solved. The psychiatrists are seemingly not interested in them.

The role played by endocrinologic therapeutics is still a minor one. The corticosteroids have not simplified the treatment of burns, but have complicated it. Meticorten and cortisones complicate the handling of the fluid

problems of burns and endanger life from disseminate infections. The only indication for their use at present is indubitable adrenal-cortical insufficiency, a very rare complication of burns.

A year ago I would have written that great advances had been made in the handling of nutritional problems of the burned; today I cannot do so. The loading of the burned person with 150 to 300 Gm. of protein daily through indwelling gastric tubes (it must be done this way because even a well person will not take so much protein) is not without danger. Ileus, diarrhea, nausea, and vomiting are minor difficulties. The protein overload syndrome of uremia, hypernatremia, hyperchloremia, hyperpyrexia, and disorientation is a lethal complication of high-protein tube feedings of the burned. The larger the amount of protein in the diet the greater is the need for water. A weak, psychologically disturbed, painful, massively bandaged, burned patient tends not to adjust the intake of water to need. Unless careful attention is paid to the osmolar concentration of body fluids while high protein feedings are forced upon the person, a life may be lost because too much protein was fed.

A re-examination of the consequences of protein depletion upon the burned animal has shown that rats bearing full-thickness burns covering 8 to 14 per cent of their bodies when fed 0.1 Gm. protein daily heal their wounds as rapidly, have no more significant infections, no more anemia, no more hypoproteinemia, and die no more often than similarly burned rats fed 8.0 Gm. of a biologically effective protein daily.

More fundamental research into the peculiarities of anabolic and catabolic alterations attendant upon thermal injury is needed before a sounder, safer approach to the handling of burn-nutritional problems can be effected.

Ninety to 150 Gm. of protein is sufficient usually to meet the protein requirements of a burned person, except under unusual circumstances, provided that the caloric deficit is not very large. The burned person expends 3,000 to 6,000 calories daily. In order that these losses be met the diet must contain a maximum amount of fat (16 to 24 per cent by weight).

The fluid balance disturbances met in the burned person are almost infinite in variety. During the first seventy-two hours postburn, functional extracellular fluid volume deficiency, water intoxication (osmolar dilution), and, in the severely oliguric, hyperkalemia constitute the main disturbances. Later hypokalemia, hypocalcemia, acidosis, and hypoproteinemia may appear. The management of all these disturbances is relatively well established.

Overzealous administration and the nonintelligent use of guide formulas constitute the main hazards of the fluid therapy of burns today. In general, fluid overloading occurs mostly among those persons with burns covering 8 to 25 per cent of the body. As shown in Table II, the majority of such burns induce circulatory changes than can be handled by the individual without the administration of saline solutions, blood, or plasma.

The burned person who demonstrates signs of shock must be actively treated even though the burn may be small, but he who does not should not be overtreated in anticipation of shock should the injury not cover more than 20 per cent of the body's surface.

An individual's physiologic recovery from a thermal injury is a difficult task in itself and recovery from the injury and overzealous fluid therapy is more difficult. It is likely that many recover from relatively small burns (less than 25 per cent) not so much because of fluid therapy but in spite of it. The same is true for larger burns but less frequently so.

The only certain way to alleviate rapidly the pain of the burned person is to give an analgesic such as morphine or Demerol intravenously (dosage one-half to two-thirds of subcutaneous dose). Subcutaneous and intramuscular absorption is unpredictably slow in the injured person.

The aseptic handling of the wound depends upon the segregation of the burned person and his wound from contaminated mattresses and mattress covers, uncovered noses and mouths, ungloved, bacterially dirty fingers, and bacteria-laden feces.

The dry massive dressing or the exposure of the wound to filtered air is usually a good means of attaining these objectives in a limited way. Perineal, gluteal, and thigh burns can be partially protected safely from bacteria-laden feces by giving Sulfasuxidine.

The practice of excising intact blisters, washing with soap, detergents, or antiseptics should be stopped. The unopen blister is a fine dressing; left alone it dries up in a week and the top comes off in eight to fourteen days, uncovering new epithelium most of the time. Detergents and effective antiseptics kill cells as well as bacteria. Should the wound be dirty, sponging and flooding with saline solution is enough.

The so-called "débridement of the burn-wound" as it was practiced was nothing more than biologic mayhem. The major mass of dead tissue was not excised! All that was done was the removal of good biologic dressings—the roofs of blisters—and the imposition of more injury upon the epithelially denuded dermis with scrubbrushes, hot saline, soap, ether, detergents, and powerful tissue poisons—the antiseptics. Let he who would commit an act upon a burned surface or put a substance upon it first apply that treatment to his conjunctiva or an experimental scald imposed upon himself. It is likely that nowhere else in the field of surgery has the dictum "prima non nocere" been more assiduously ignored than in the primary care of the burn-wound.

The relative efficacy of simply exposing the burn-wound to air is likely relatable, in part, to the cessation of detrimental "therapeutic" acts. The application of antiseptics to amputation stumps before closure and the washing of the peritoneal cavity with antiseptics as treatment for peritonitis were discontinued thirty years ago; it is high time that the burn-wound belatedly received the same consideration as the inflamed peritoneum and amputational flaps. Who among us would wash an inflamed, bacterially laden peritoneal cavity with Septisol, green soap, ether, or alcohol, or apply to it salves containing Furacin, phenol, acriflavine and so on? Fundamentally, the burn-wound differs little from an inflamed contaminated peritoneum.

The handling of an infected burn-wound is still somewhat primitive. The infrequently changed wet-dressing covered with an impervious material is a

bacterial incubator. The infected wound covered with gauze thickly coated with grease is an abscess the outer wall of which is the grease-gauze. When these dressings are removed there is poured, sprayed, or dabbed upon the wound cell-breaking detergents such as liquid soap or Septisol; powerful protein denaturants such as alcohol and ether; epithelial growth inhibitors such as scarlet red; general biologic cellular poisons (antiseptics) such as Furacin, Mercurochrome, Mercresin, tincture of iodine; and another bacterial poultice or abscess wall is then reapplied. No wonder that simple washing of the wound with saline solution and exposure to even bacteria-containing air has returned as a superior method of treating the infected burn-wound after a lapse of fifty years.[2]

Nontraumatic mechanical absorptive cleansing of dirty or crusted infected burn wounds may be effected also by the frequent changing (q. one to 3 hours) of thick, uncovered, saline-wet, lay-on dressings. Capillarity works to pull bacteria from the wound into the wet dressing for one to three hours and the repeated application and removal of such a dressing reduces the bacterial population on the wound.

The excision of a dry eschar with the knife is an improvement upon the uncontrolled bacterial digestion of the wet eschar. The latter has been practiced too long. The infrequently changed massive occlusive dressings interspersed with tub-soaking and the piecemeal snipping and tearing off of wet, gangrenous skin abrogates all surgical principles. The significance of the reduction in morbidity and mortality attendant upon surgical excision of the dry eschar in Military Burn Centers can no longer be ignored.

The advances in graft coverage of thermally denuded surfaces have been few. The electrically driven dermatome is so easy to handle that it has become a hazard. With it donor areas are so smoothly and deeply denuded that many of them become massive, open, epithelially denuded wounds; injury is added to injury. No machine yet made thinks or feels and the taking of grafts is still an art to be practiced by experienced, or at least intelligently directed, hands.

Split-thickness homografts taken from the bodies of the briefly dead and applied immediately or after periods of storage up to twenty days, have proved to be remarkably efficacious as temporary, self-attached biologic dressings that reduce the loss of fluid, the wound-area subject to infection, and fever. Staged autograft coverage of very large defects is rendered easier with their use. In addition, they also reduce the magnitude of the nutritional problems attending large third degree burns.[3]

I, too, would like to close the discussion with a quotation from Mont Reid's classic paper which was quoted by Dr. Sumner L. Koch.[4, 5] Many eyes may have missed it that might see it now.

> Pasteur's work told the world what was causing the infection of wounds and unloosed . . . a veritable flood of bactericidal agents which show no signs of abating even today. Paré astounded himself and the surgical profession of his time when he learned that the sloughing incident to the burning of wounds with boiling oil or the actual cautery had a very deleterious effect upon the healing of

wounds. He learned that the gentlest cleansing of the wounds with the commonest innocuous substances at his disposal at that time, such as water and soap, gave the best wound healing. Today, with our knowledge of the delicacy of growing living cells and of their lethal susceptibility to alcohol, ether, iodine, Mercurochrome, Merthiolate, carbolic acid, bichloride of mercury, and countless other substances, people go blithely on pouring them into open fresh wounds to kill living cells and to complicate the process of wound healing and to promote the chances of infection. So far as fresh traumatic wounds are concerned we would today be far better off with a total ignorance of all chemical bactericidal agents and if we only utilized our knowledge of bacteria and of wound healing by gentle mechanical cleansing of all the surrounding skin and open wound of all dirt, foreign bodies, dead or devitalized tissue and by flooding the invisible bacteria away by means of sterile salt solution. Paré was, in the light of modern knowledge, densely ignorant, but he acted with amazing sanity; today, with a colossal knowledge of the properties of both bacteria and living cells, many wounds are being treated with a lack of sound judgment.

REFERENCES

1. Allen, G.: Personal communication.
2. Sneve, H.: J. A. M. A. **45**: 1, 1905.
3. Brown, B.: Personal communication.
4. Koch, S. L.: SURGERY **38**: 452, 1955.
5. Reid, M.: New England J. Med. **215**: 753-756, 1936.

Book Reviews

Bone, an Introduction to Skeletal Tissue. By McLean and Urist. Pp. 182. Chicago, 1954, University of Chicago Press.

This small monograph by a distinguished student of bone physiology and an active clinical orthopedist is an ideal example of what can be accomplished by such collaboration. Rather than a historical review of the literature, this is an up-to-date survey of present concepts in the field of bone physiology. It presents in brief and concise form what is believed today in regard to the complex functions of bone as a supporting tissue and as an actively metabolizing tissue. The authors have done an outstanding job in making available to orthopedists the basic information which they need to understand about what is going on in their patients. This book can be read with profit by any student of bone physiology.

Fractures in Children. Walter P. Blount. Pp. 279. $9.50. Baltimore, 1954, Williams & Wilkins Company.

This book has been a long time in preparation and it is very fitting that its publication coincides with the term of its author as president of the American Academy of Orthopedic Surgeons. Dr. Blount has lectured tirelessly throughout the country preaching conservatism in the treatment of fractures in children. Any one of those who have had the opportunity to hear his delightful talk will not be disappointed in this book.

One feature which makes it especially outstanding is the x-rays of the long-term follow-up of the cases presented which beautifully demonstrate the effect of the normal molding of the growth process upon the deformities produced by fractures.

Another feature of this book which deserves special notice is the fine pen drawings of Mr. Carl Brill which add immeasurably to the text. There are several appropriate cartoons which emphasize the most common errors made in the treatment of fractures in children.

Orthopedists will need no urging to add this book to their library since many of them have been waiting anxiously for a long time for its appearance.

The general physician who treats fractures in children can be well advised to provide himself with this monograph because it is primarily designed to answer his questions and to provide suggestions and solutions to the many problems which arise in the treatment of fractures in children. The publishers have done very well by the author and have produced a book which is easy to read and nicely laid out.

Hernia: The Pathologic Anatomy and Their Anatomic Repair of the More Common Hernias. By Chester B. McVay, University of South Dakota. Pp. 40. Springfield, 1954, Charles C Thomas, Publisher.

This monograph in the Pictorial Surgical Technique Series covers diaphragmatic, epigastric, umbilical, inguinal, femoral, incisional, lumbar, and pelvic hernias. The text is brief and is tied intimately to the 19 excellent illustrations, and the text for each illustration appears on either the same or the facing page.

The anatomic derangements and surgical repairs of inguinal and femoral hernias comprise one-half of the monograph, and the author's techniques of "abdominal inguinal ring repair" and "reconstruction of the posterior inguinal wall" are illustrated and described.

Hernias other than inguinal and femoral hernias are covered adequately, but the excellence of the section on the inguinofemoral region makes this monograph outstanding.

An Atlas of Operations for Trauma. By Otto Russe. Pp. 106. Wien-Bonn, 1954, Wilhelm Maudrich.

This is an atlas of procedures to be used in the treatment of patients with acute traumatic injuries and during the reconstructive phase. It is presented in the form of an atlas of illustrations, with text in English, French, and German. A wide variety of subjects is covered, including intestinal anastomosis, operations for subdural hematoma, and techniques for skin grafting.

The book has only 106 pages so it can be seen that no subject is covered thoroughly. Perhaps the most valuable portions of the book are those dealing with anatomic exposures of the bones and joints. The book is valuable chiefly as it suggests various possibilities in managing a wide variety of injuries. Some of the procedures described are standard methods widely used in this country. Others will be somewhat unfamiliar to American surgeons.

This would be a good book to have in the operating room, particularly so that members of the resident staff could browse through it occasionally and familiarize themselves with the wide variety of methods which can be applied in the treatment of accident cases.

Die Chirurgie Des Ellbogengelenkes. By Otto Wustmann. Pp. 332, with 118 illustrations. Berlin, 1954, Walter de Gruyter & Co.

This is a pedestrian catalogue of all the conditions which can involve the elbow joint. It suffers the handicap of all regional books which discuss generalized diseases affecting that particular region, such as tuberculosis, osteomyelitis, arthritis, and tumors. The major operative approaches to the elbow joint are described but not illustrated. In the same regard, many of the illustrations are remarkably uninformative. It is doubtful that this book will be useful even to those surgeons who can read German easily.

Die Aseptischen Chondro-Osteonekrosen. By O. Häuptli. Pp. 233. Berlin, 1954, Walter de Gruyter & Co.

This book is a thorough review of the literature dealing with the large group of conditions which include Legg-Perthes disease, Calvé's disease, Osgood-Schlatter disease, Kienböck's disease, and so on. It is a brief review of all of the histologic material obtained from human specimens and of all of the experimental work which has been done to clarify these conditions. This excellent summary brings up to date all of the information which we have about this puzzling group of conditions. It is especially valuable because it is brief and to the point. It is not padded. The bibliography is complete and unobtrusive. This is a valuable book for anyone studying these conditions.

An Atlas of Musculoskeletal Exposures. By H. F. Moseley. Pp. 235. Philadelphia and Montreal, 1954, J. B. Lippincott Company.

The outstanding feature of this book is the beautiful color reproductions of the anatomic drawings. Accompanying these beautiful drawings is a short explanatory text. In addition to this descriptive material there is a discussion of the positioning and draping of the patient for an operation upon the area to be described. While the description of the positioning of the patient for the operative procedure is of value, the photographs illustrating the procedure of draping leave much to be desired and, in most cases, do not fully convey the technique used by the author. It would also appear that the description, in regard to skin preparation and operative techniques generally, is superfluous baggage in a book which otherwise brilliantly conveys the essential information regarding the surgical anatomy.

One especially nice feature of the book is the bibliography given at the end of each section which includes, among other things, a list of motion pictures demonstrating these surgical approaches and the sources from which these films can be obtained.

This is a very creditable contribution to the already extensive literature on musculoskeletal exposures.

Living Bone in Health and Disease. By Irvin Stein, Raymond O. Stein, and Martin L. Beller. Pp. 510. Philadelphia, 1954, J. B. Lippincott Company.

This book is a well-illustrated, well-printed potpourri of information about the bone and health and disease. It is not an encyclopedia containing information on all bone diseases. Exactly why the authors chose to discuss some diseases and omit others is not clear. However, the topics which are discussed are discussed fully, with a complete review of the present status of our information regarding the conditions. The continuous reference to the "senior author" seems unnecessary in a book which is presented to the reader as a collaborative effort. The lengthy bibliographies at the end of each chapter lose a tremendous amount of value because they are not presented in alphabetical order. It is necessary to find the reference number in the text before papers by specific authors can be located easily. This is a tremendous oversight on the part of the editor. This book will be a valuable primer for those physicians making their first acquaintance with the literature on bone and bone diseases. For the advanced student of these conditions, this book will be of little, if any, value.

Tumors of the Soft Tissues. By A. P. Stout. Monograph, Armed Forces Institute of Pathology, Washington, D. C., 1953.

The author has had tremendous experience with tumors of the soft tissues. He emphasizes the difficulty of differentiating between the various types of soft-tissue tumors and that in many there may be multiple elements making it difficult to designate the particular type of tumor. It is of importance to differentiate between malignant and benign soft-tissue tumors. The present report is based upon a study of 7,337 benign mesenchymal tumors observed in the Surgical Pathology Laboratory at Columbia University and 1,349 malignant mesenchymal tumors.

The various benign and malignant tumors are discussed and excellent and profuse illustrations, many of which are in color, are presented. The final chapter on diagnosis and treatment is excellent. The author emphasizes the importance of accurately determining the type of tumor before therapy is undertaken and believes that this should not be made on a frozen section. Only in this way can the physician accurately determine the best type of therapy. There are some malignant lesions which require very radical procedures, others which require locally extensive removal, and others which are best treated by irradiation. This monograph should be in the library of every pathologist and surgeon.

Tumors of the Major Salivary Glands. By F. W. Foote, Jr., and E. L. Frazell. Monograph, Armed Forces Institute of Pathology, Washington, D. C., 1954.

As is typical of the other fascicles of the Atlas of Tumor Pathology, this one is profusely and excellently illustrated. The study is based upon an analysis of 877 cases of major salivary gland tumors observed at the Memorial Hospital, of which 494 were benign mixed tumors, 57 malignant tumors, 51 low-grade mucoepidermoid tumors, and 47 high-grade mucoepidermoid tumors. In addition to these, squamous-cell carcinomas, adenocarcinomas, acinic-cell tumors, and papillary cystadenomata lymphomatosa were considered as well as the rarer type. In each instance, a brief historical review is given, then the gross and microscopic description accompanied by excellent photographs and photomicrographs, and the natural history of the disease. Anyone interested in surgery of the neck or in pathology of the tumors of the major salivary glands should have this fascicle.

Tumors of the Retroperitoneum, Mesentery, and Peritoneum. By L. V. Ackerman. Monograph, Armed Forces Institute of Pathology, Washington, D. C., 1954.

Although this fascicle covers a large number of entities, it is excellently done. Benign and malignant tumors arising in the retroperitoneal space from adipose tissue, smooth muscle,

connective tissue, striated muscle, lymph vessels, blood vessels, primitive mesenchyma, all of which are of mesodermal origin, are discussed; and the tumors of neurogenous origin are discussed. There is also a discussion of the tumors of remnants of renal blastoma and tumors arising from embryonic remnants, particularly the benign and malignant teratomas and the chordomas; and finally, metastatic malignant tumors, particularly those of the peritoneum. This fascicle is profusely illustrated and the text is concise and lucid. It is extremely difficult to cover such a wide field, but it is excellently done. A particularly valuable chapter is that on the discussion of chordoma.

Tumors of the Stomach. By A. P. Stout. Monograph, Armed Forces Institute of Pathology, Washington, D. C., 1953.

This monograph is based upon a series of 110 benign tumors and 541 malignant tumors studied in the Laboratory of Surgical Pathology of Columbia University. The author emphasizes the relatively low incidence of benign tumors in the stomach and the fact that most of the malignant tumors are carcinoma. Of the benign tumors, adenomatous polyp and leiomyoma occupied first place. Of the malignant tumors, carcinoma, leiomyosarcoma, and lymphosarcoma occupied first place. The other types are relatively rare.

A general consideration of the cause of gastric tumors is discussed, following which there is a brief discussion of gastritis and then a discussion of the benign and a very elaborate discussion of the malignant tumors, particularly carcinoma. This fascicle, as are the others, is elaborately and excellently illustrated, many of the illustrations being in color. The various types of carcinomas are described and their clinical course is emphasized. Twenty-two and five-tenths were found to be fungating, 27.5 were penetrating, 8.3 per cent superficially spreading, 4.5 per cent were of the linitis plastica type, and there were 37 per cent in which there was no special type. The type of spreading in all types of carcinoma is carefully described and this is of extreme importance to the surgeon. Differentiation between carcinoma of the stomach and other lesions which simulate it is also given. Anyone interested in surgery of the stomach or in pathology of the stomach should have this monograph.